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THE FREE TRANSPLANTATION OF SKIN

AN EVALUATION OF METHODS[1]

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THE transplantation of skin by means of pedicled flaps was a well known practice centuries before the development of early European surgery. Among the earliest procedures are those known as the "Indian" method, and the "Italian" method, both of which were used principally in the restoration of the nose or other parts of the face which had been amputated by enemy peoples. Although countless modifications of these principles were made over a period of many years, one of the most important improvements in the transplantation of skin by the pedicle method was developed by Gillies (10), who, after undermining between parallel incisions, rolled this skin into a tube, held so by suture of the edges. This principle was described by Filatoff prior to Gillies' publication of the method, but has become known since as the Gillies tube method of skin transplantation and together with the Indian and Italian methods, has been proved to be a sound practical method of pedunculated skin transplant.

In contrast to the pedicle principle, the free transplantation of skin implies the excision of a portion of skin and transference of this graft to another portion of the body, its viability depending upon immediate fixation to the underlying tissue and the rapid establishment of a capillary blood supply. The development of this method of skin transplantation belongs to a relatively modern period in comparison with the antiquity of the pedicle flap types.

Because of the tendency for most writers unconsciously to favor the particular type of skin graft with the development of which they have been concerned, it is perhaps justifiable for one who has fostered no new ideas relative to the subject, but who has tried to profit by the best in each, to criticize the most common present day methods of free skin grafts.

Reverdin is generally credited with the first successful demonstration of the transplantation of small bits of skin from a remote part of the body applied to a granulating surface. Epithelization of the defect was seen to follow the application of these small grafts which were epithelial islands, proliferating at the borders and ultimately coalescing. In addition to the importance of Reverdin's work as being the first practical method of free skin grafting, it is of considerable interest that his favorable results were obtained before the era of antiseptic and aseptic surgery. First thought to be grafts of epidermis only, Reverdin later recognized that his grafts contained a portion of the corium.

Davis (5) in 1914 first described the "small deep graft" and has reported its use in several communications since that time (6, 7). In principle, there is little to differentiate it from the "pinch grafts" of Reverdin except that a

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greater portion of corium is removed with the central portion of the Davis graft, and further that the bit of skin to be removed is raised with a needle rather than being "pinched" upward into a tented contour by forceps. The grafts are placed at some distance from each other upon a granulating area, and, in both the Reverdin and Davis procedures, healing depends upon the eventual coalition of these small epithelial islands.

The value of this type of graft is indisputable for the repair of large defects where the donor areas are relatively limited and where safety to the patient demands a minor surgical procedure. Because of the simplicity of the method and the assurance of a high percentage of "takes" even upon grossly infected granulations, these small grafts have become widely popular and each year a great many burned children are rehabilitated by the use of this type of graft only.

There are, however, several definite objections common both to the pinch graft and the deeper graft of the same size, among the most important of which is the appearance of the grafted area after healing is complete (Fig. 1). Because the central portions of the grafts include a thickness of corium not present at the edges nor in the spaces between the grafts, an uneven, mottled surface results, which has an unpleasant appearance, particularly on exposed body surfaces. The same objection applies to the donor areas, which are usually the thighs, and which have an unsightly appearance after healing has occurred.

Owing to the fact that these small grafts are generally placed upon old granulations and that some time must elapse before they proliferate sufficiently to cover the defect, it is inevitable that some of this granulation tissue must remain permanently as undesirable scar, under and between the small epithelial islands. Needless to say, if there is a tendency toward keloid formation, the use of this type of graft is highly unsatisfactory.

After an experience of several years, Thiersch in 1886 reported on a method for the grafting of large sheets of partial thickness of skin which included a portion of the corium. Although Lawson and Ollier had both used this method prior to the work of Thiersch, the latter's name is usually applied to this type of graft. It marked a distinct advance over the small "pinch" grafts of Reverdin, since much larger areas were covered in one procedure, and it is probable that this type of free skin graft has enjoyed a greater popularity and served a broader field of usefulness than any other to a most recent period.

These larger thin split sheets of skin have, in addition to many of the desirable features of the small grafts of Reverdin and Davis, relatively few objectionable characteristics. Where adequate donor areas are available, sizeable defects may be covered in one operation with a fair expectancy of "take" even upon an infected surface. Because of the greater size of the individual grafts, which vary depending upon the donor areas available and the dexterity of the operator, there is less chance for the formation of scar between grafts than in the smaller variety, and the final appearance of several large sheets of grafts is usually better than that of an area covered by a great many small coalescing islands of epithelium. If care is exercised in cutting Thiersch grafts not to incise into the subcutaneous fat, spontaneous regeneration of the entire donor site will rapidly take place and the area will eventually assume the appearance and texture of the surrounding skin. Reference to this regeneration will be made later.

It should be mentioned, however, in a fair criticism of the Thiersch graft, that it includes, in most cases, only a small portion of the corium removed with the epidermis which at best provides a poor foundation for epithelium, and in areas subject to pressure or friction, may provide quite inadequate protection for the underlying tissue. Considering also the great thickness of elastic corium in normal skin, it may also be anticipated that considerable contraction will occur under a graft having for its anchorage the thin portion of corium usually found in a Thiersch graft.

The first use of a full thickness free skin graft is generally conceded to Wolfe of Glasgow who, in 1875, reported the repair of a defect about the eye by this method. Although other workers undoubtedly deserve credit for the development of this type of fat free, full

thickness graft, it is usually designated as the "Wolfe graft."

For appearance, durability, and in resistance to contracture, there should be perhaps, no free skin transplant as satisfactory as the Wolfe graft. Containing as it does the entire thickness of corium with the epidermis, it provides a satisfactory covering for defects in practically any location, including the hands and feet. Because the edges of the graft are accurately approximated to the corresponding edges of the defect, a linear scar only separates the two, and, if there has been adequate excision of scar tissue, contracture will be reduced to a minimum.

Certain definite conditions are essential, however, for the successful use of the free full thickness graft which restrict its use. It requires, first, that the base upon which the graft is placed be uninfected, which eliminates the common granulating areas which demand covering. The actual removal of the graft and the application to its new location requires meticulous care with attention to tension, provision for the escape of serum, and pressure. In addition, because a full thickness of skin has been removed, the donor area requires closure by some method, constituting, in the case of a large graft, an additional problem.

Considering the indications for its use, as well as the technical requirements, it is possible that the Wolfe graft has, properly, a relatively limited field of usefulness, and will remain a more specialized type of graft than those others under consideration in this paper.

Tunnel grafts, or buried skin grafts, were described by MacLennan in 1912, Moszkowicz in 1916, and Esser in 1917. The method comprises the introduction of full thickness pieces of skin upon (or without) a mold beneath granulations or scar. This is usually done by means of parallel incisions on either side of the area to be grafted, and several days later, when the grafts have become attached in their buried bed, the overlying scar or granulations are cut through and the grafts exposed. Parce has been given the credit for the origination of this type of graft by Col. Keller (12), who reported an extensive experience with it over a 10 year period.

Fig. 1. Extensive burn treated by small grafts of Reverdin or Davis type. The entire area is covered by a thick keloid growth, with relatively few small areas of good skin. The poor result in this case was due in part to an inadequate number of grafts, and in part to the formation of keloid, but illustrates the generally poor cosmetic end-result obtained with this type of graft.

The buried type or tunnel grafts have, in addition to some undesirable features, many to recommend their use. Of decided merit is the fact that this, like the Wolfe graft, is of full thickness skin and should therefore approach the ideal for the covering of defects over bony prominences and in areas subject to pressure. In addition, the "tunnels" made to receive these grafts may be made beneath either scar or granulation tissue, and therefore provide a method for a full thickness, free skin transplant even in the presence of an infected surface.

As generally employed, however, they are rather small grafts and not particularly suitable for large defects. In addition, the cosmetic end-result is only fair, and the donor area, as in the case of the Wolfe graft, requires

a.

b.

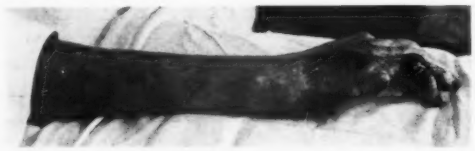
Fig. 2. a, Removal of a thick split graft from the thigh. The suction retractor designed by Blair precedes the course of the knife and maintains an even tension of the donor area. The tongue blade, held by an assistant, should be advanced behind the knife when large grafts are to be removed. b, A sizeable graft of uniform thickness ready for removal and application to the denuded area. A larger graft could have been removed without difficulty, but measurement of the defect made previously showed it to be adequate.

closure, since the entire thickness of skin has been removed.

For a number of years, Blair and Brown (1) have been advocating the use of large, free, split-thickness grafts in the repair of extensive cutaneous defects. They have suggested the term "thick split graft" (since it includes one-half to three-quarters of the thickness of skin) to differentiate it from the thinner Ollier-Thiersch graft and also from the full thickness Wolfe graft. Their technique for the removal and application of these grafts has been adequately described in several publications (1, 2, 3, 4), and requires no amplification. In principle, these thick split grafts are quite similar to Ollier-Thiersch grafts, except that very much larger individual grafts are cut, the area to be grafted is specially prepared, and the application and fixation of the grafts differs from that commonly employed in the use of the Ollier-Thiersch grafts.

These large split thickness grafts have, according to Blair and Brown, more of the good than the bad features of either the full thickness graft or the thinner Ollier-Thiersch graft. The writer's experience of several years unqualifiedly supports their opinion.

By the use of a suction retractor, and a specially constructed knife described by Blair (1) in 1929, large individual grafts of uniform thickness 25 to 50 square inches may be cut from suitable areas (Fig. 2). Often a single graft is sufficient to cover the defect and if not, the adjacent edges of the grafts are su-



a.

b.

c.

Fig. 3. a, Gasoline burn of 6 months' duration. The portion of the hand not denuded is covered by a thin and quite unserviceable epithelium on a scar base. The graft on the forearm was done 3 weeks prior to this photograph. b, Excision of the scar and granulations down to tendon sheaths by sharp dissection. The bleeding is usually controlled temporarily by hot packs and pressure, although the application of the graft acts as an excellent hemostatic. c, Application of the graft. Allowance is made for some overlapping of the graft at the edges of the defects and accurate approximation is effected by a continuous mattress suture of fine silk or some comparable material. Multiple perforations are made in the graft to permit the escape of serum and blood, and a pressure dressing is applied after the careful removal of clots from beneath the graft. Although unnecessary in this instance, pressure over surfaces on the trunk or in situations such as the axilla is best maintained by the use of large sea sponges which are moistened before they are used.

a.

b.

Fig. 4. a, Appearance of the hand shown in Figure 3, 3 weeks after grafting. The careful suturing of the graft edges produces only a minimal linear scar which becomes almost obliterated in time. Motion of the fingers is limited by scar and damage to joints themselves, but the tendons move freely without fixation under the graft. b, Volar aspect of the same extremity. The single graft on the forearm was done 5 weeks prior to this photograph.

tured together, producing only a linear scar. Because of the size of these grafts, surfaces 100 square inches or more may easily be covered in one procedure.

The thickness of the grafts varies somewhat, depending upon the age and sex of the patient, as well as the location of the donor area, but in general, one-third to three-quarters of the thickness of the corium is included with the epidermis. This in most cases, is a depth great enough to provide the graft with accessory skin structures, and the end-result is a durable covering containing hairs, sebaceous and sweat glands, and closely resembles full thickness skin. Although this graft may prove impractical in a location subject to great friction or actual weight bearing, it may be of great value as a temporary covering for such a granulating area, preliminary to an eventual full thickness graft of some variety.

Preparation of a granulating area to be grafted includes, in addition to the usual measures to minimize infection, (1), excision of the scarred border of the defect, and (2), slicing off of the superficial edematous granulations together with some of the underlying scar until a firm scar base is reached (Fig. 3). This preparation, together with adequate provision for drainage by perforations in the graft, and careful fixation of the graft with suture and a pressure dressing, by means of a marine sponge, assures one of almost 100 per cent "take" in the great majority of cases. The first postoperative dressing may be done at any time after 3 or 4 days when the grafts will usually be found to be firmly grown to the underlying scar and to require little or no further attention (Fig. 4).

Healing of the donor areas from which these large thick split grafts are removed, is of some interest, since if one is careful to avoid cutting into the subcutaneous fat, spontaneous regeneration of even very large areas will occur within a week to 10 days. Covering of these areas with a tannic acid crust is a satisfactory dressing though probably less comfortable than a firmly applied gauze dressing left unchanged for 7 to 10 days.

Regeneration of an intact epidermis after the removal of an Ollier-Thiersch graft was formerly thought to be from the deepest portions of the papillary layer of the epidermis left below the level at which the graft was taken. With the demonstration that thick split grafts included in addition to the entire epidermis, enough corium to remove hair bulbs situated deep in the true skin, it was apparent that an entirely new epidermis must regenerate from structures in the remaining thinned-out dermis (Fig. 5). Biopsies removed from the donor site on a thigh at the time of removal of the graft, and 3, 6, and 10 days later, show without question, that the regeneration of the new epidermis is from the hair follicles and associated sebaceous glands, and that within the 10 day period, a complete new epidermis has been formed, differing in some respects from the previous one but having very active growth and well differentiated cellular layers (Figs. 6, 7, 8, 9).

This mode of regeneration is not surprising when one considers that the hair follicles are embryologically invaginations of epidermis and that the outer root sheath of the follicle is a direct continuation of the stratum germinativum of the epidermis. Since it is also

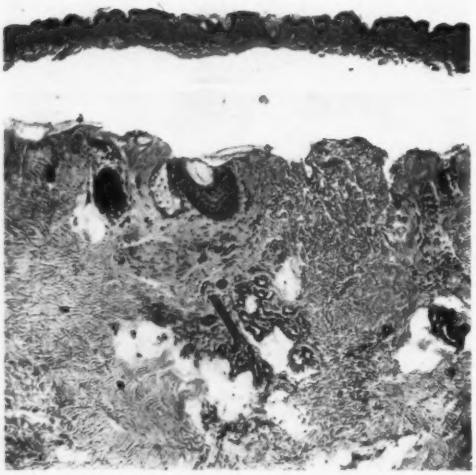


Fig. 5.

Fig. 6.

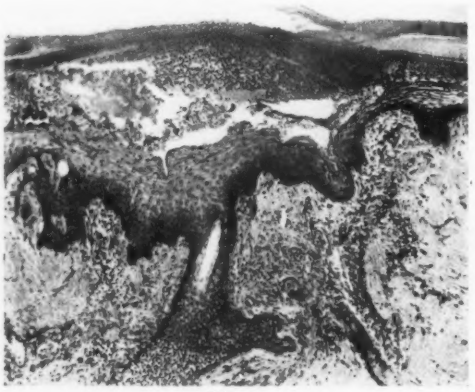


Fig. 7.

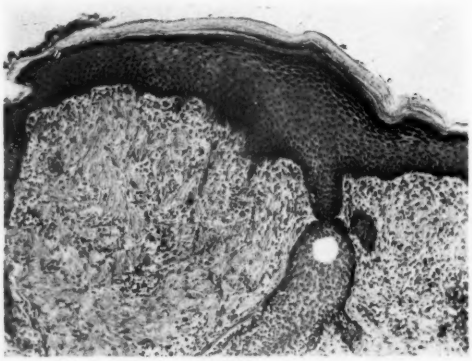


Fig. 8.

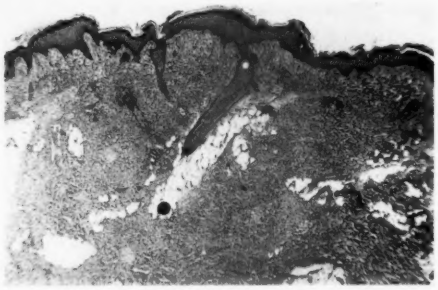


Fig. 9.

Fig. 5. Low power photomicrograph of thick split graft (above) and higher power of a section of the remaining corium and subcutaneous tissue, removed immediately after the graft was cut. The graft includes, in addition to the entire epidermis, an appreciable portion of corium, although less than is usually removed. Note the obliquely cut hair follicle on the denuded surface of the donor area.

Fig. 6. Biopsy of donor site 3 days after removal of thick split graft. Photomicrograph of section cut at right angles to the denuded surface, passing obliquely through two hair follicles. A single layer of columnar cells, deeply stained and continuous with the outer root sheath of the hair follicle, is seen extending over the surface of the corium. Immediately above the mouths of the follicles, additional layers are seen which have become stratified, squamous epithelium. A marked inflammatory reaction is seen throughout the corium.

Fig. 7. Biopsy of the donor area immediately adjacent to that shown in Figure 6, 6 days after removal of graft. The source of the new stratum germinativum from the outer root sheath of the hair follicle is more clearly shown, with an exaggerated thickness of the new epidermis in the immediate vicinity of the follicle. The stratified squamous character of the upper layers is seen, with beginning cornification and desquamation of the surface layers beneath the blood and debris.

Fig. 8. Section of biopsy taken from the area adjacent to that shown in Figures 6 and 7, 10 days after removal of graft. An entirely new epidermis has regenerated over the surface of the corium, having more mature characteristics and greatest thickness near the hair follicle.

a. b.

Fig. 10. a, Burns of hands 19 years previously. X-ray treatments for scar resulted in radiodermatitis with carcinomatous change on the dorsum of both hands. The lesion on the right had been excised and the defect repaired by a pedicle flap. We excised a carcinoma 3.5 centimeters in diameter from the left hand and used a thick split graft for repair. There was no recurrence or metastasis in 1½ years. We favor, as does the patient, the end-result on the left hand from the standpoint of appearance, function, and loss of time involved. b, Function of both hands equally satisfactory.

true that the actual number of hairs may increase during life by the development of new follicles after the manner of the primary formation (18), it is logical to expect that with the traumatic or surgical loss of the epidermis, the process will be reversed, with an upward growth of the outer root sheath of the hair follicles in the region and a fusion of these cellular outgrowths to form a new epidermis. This conception of epidermal repair may have some relation to the almost universal distribution of hair over the body surface and accentuation, with reference both to the number of hairs and coarse nature, over body surfaces commonly subject to trauma, such as the dorsum of the hands and arms, legs and anterior aspect of the trunk. The rapid regeneration of a new epidermis from hair follicle proliferation seems less remarkable when one considers the unusual growth properties of hair in general, and particularly the beard of an adult male. There are probably few structures in the body capable of as active growth as that shown by hair.

The thick split graft has then, in addition to others, two decided advantages as compared with other types of free grafts since (1) the graft itself has many histological characteristics and the gross appearance of full thickness skin and (2) the large areas from which the grafts are cut regenerate spontaneously with a modified, but thoroughly serviceable, type of epithelium.

Fig. 9. Low power photomicrograph of same section as in Figure 8. A comparison of the new epidermis with that removed with the graft shown in Figure 5 (same magnification) demonstrates a striking difference. The new is several times thicker, due probably to the demand for epidermal repair, or possibly because of the relative thinning of the corium a portion of which was removed with the graft.

SUMMARY

For the reasons enumerated in the discussion of the pinch and the small deep graft, we have restricted its use to cases, usually children with extensive burns, in which the available donor areas are relatively limited and in which the poor condition of the patient permits only a minor surgical procedure to initiate repair. In this group of cases, there is no doubt of its usefulness, admitting at the same time its shortcomings.

Although the Wolfe, or full thickness free graft should be, theoretically, an ideal type of skin transplant in many instances, we must agree with a recent statement by Gillies (11), that "the standard of technique for a successful result demanded by this type of repair and its tendency to subsequent mild trophic disturbance, do not justify its use in place of more certain methods."

With reference to the tunnel or buried full thickness graft, the rather narrow scope of applicability has limited and probably will continue to limit its use with a corresponding popularity of other types.

Although it has been generally felt that the repair of defects in certain locations such as around joints or over movable structures as tendons, require full thickness skin, we have found that the thick split grafts furnish an adequate covering in many instances, and reduce substantially the long periods of hospitalization necessary for the use of full thickness free, pedicle, or flap types of grafts (Fig. 10).

In commenting upon the thick split graft, it should be mentioned that the successful removal of large grafts requires some degree of manual dexterity and above all a sharp knife. With practice, however, and the meticulous care of the knife that one gives to a razor, surprisingly large grafts of uniform thickness may be removed easily and quickly. We feel that the suction retractors designed by Blair have been of inestimable help to us in cutting these grafts.

Several factors mentioned in the previous parts of this paper have influenced the trend of free skin grafting in our hands, resulting in a much wider use of the thick split graft and a corresponding infrequent use of the Ollier-Thiersch and the full thickness types of grafts. There are relatively few instances in which a very thin Thiersch graft is actually preferable to the thicker split graft, one of these being in lining the orbit. Since the donor area heals with equal readiness, regardless of the surface area or the thickness of the graft, provided the knife passes continuously *through* rather than *beneath* the corium, our preference has shifted decidedly in favor of the thicker one. Most situations require, for the ideal, a greater thickness of corium than is usually included with either type, which recommends in many instances the thickest graft which can be cut without the removal of full thickness skin.

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BILIARY DYSSYNERGIA: PHYSIOLOGICAL OBSTRUCTION OF THE COMMON BILE DUCT

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DILATATION of the bile ducts and intrahepatic biliary radicals indicates an obstruction to the free flow of bile from the liver into the duodenum. Calculi, inflammatory reactions, strictures, neoplasms, kinks, and functional derangements of the choledocho-duodenal-sphincteric mechanism are the most important obstructive agents. Considerable emphasis has always been placed on the organic obstructions of the common duct, but little attention has been paid to the physiological dysfunctions of the choledochal sphincter which are capable of producing an enormous dilatation of the entire biliary tract.

Many surgeons have encountered hepatic congestion, bile stasis, and dilatation of the "biliary tree" without being able to demonstrate pathological changes to account for the obstruction. Such was the experience of Krukenberg who, in 1903, reported a case of "gall-stone colic" in which neither calculus nor infection was found. In 1909, Aschoff and Bacmeister described the "stasis gall bladder" which occurred in the absence of stones, inflammations, strictures, or kinks. As a result of physiological experiments which were predicated on the anatomical work of Oddi, Meltzer in 1917 suggested that a spasm or an increased tonus of the sphincter of Oddi might act as a mechanical impediment and produce biliary colic and icterus.

Schmieden, in 1920, offered a new idea. He had, in several instances, encountered markedly distended, but otherwise normal appearing, gall bladders in which he felt the distention might be caused by a kink or anatomical defect at the junction of the cystic duct with the gall bladder. According to Ivy, this theory was quickly accepted but was later criticized because the hypothetical anatomical hindrance could not always be demonstrated.

In 1922, Berg continued the studies of Meltzer and Westphal and concluded that a functional derangement, particularly a spasm, of the sphincter of Oddi could easily produce a chronic stasis of bile. Newman, Nuboer, Giordano and Mann, each reported cases in which a hypertrophied sphincter of Oddi had resulted in an occlusion of the common bile duct. In 1933, Strauss et al. presented 22 cases of chronic bile retention in which duodenitis with inflammation of the ampulla of Vater seemed to be the sole cause of obstruction. During 1934, Ivy and Sandblom made an intensive study of the abnormal motor responses of the gall bladder and bile ducts. They concluded that a functional disorder of the choledochus resulted in the accumulation of bile. To differentiate this from other forms of bile stasis, they used the term "biliary dyskinesia," which we shall designate as biliary dyssynergia, a more descriptive term.

By means of direct roentgenographic visualization of the biliary tract with radiopaque oils, we have been able to demonstrate that an increased tonus or spasticity of the choledocho-duodenal-sphincteric mechanism is capable of producing a mechanical obstruction, thus causing a retention of bile. These deductions were corroborated by operative exploration and are predicated on the following experimental investigation.

CASE 1. Mrs. O. M., housewife, aged 58 years, entered the hospital complaining of "heartburn." During the past 10 years she had suffered from recurrent attacks of indigestion, characterized by epigastric distress, belching of gas, passage of flatus, and abdominal cramps. The ingestion of fatty foods, cabbage, apples, or pork always initiated an attack. In the last 4 years she had had several paroxysms of gall-stone colic without jaundice.

The patient appeared to be a well nourished woman. The gall bladder could not be palpated but there was slight tenderness in the right subcostal area. The gall bladder concentrated the cholecystographic dye but it did not empty well following the fat meal. Several small circular shadows were seen which were thought to be gall stones. Jaundice was not a factor, the icteric index and van den Bergh reactions being normal.

Fig. 1. Case 1. Note the marked dilatation of the bile ducts. An obstruction near the ampulla has prevented the lipoiodine from entering the duodenum. Radiograms made 3 hours later indicated the pseudo-obstruction had disappeared for the lipoiodine was then in the jejunum. A spasm of the choledochal sphincter followed by a relaxation offers a probable explanation for this observation. (Unfortunately the 3 hour plate was destroyed.)

At operation the liver presented no abnormalities except a mild fibrosis of its capsule. The gall bladder was distended and contained several small stones. Its walls were thick, firm, and indurated. The cystic duct was small, pliable, and contained no calculi, but it was sharply kinked at its junction with the gall bladder. The walls of the common duct were slightly thickened but there was no evidence of dilatation, stones, or neoplasm. The pancreas was of normal size and consistency. Black, viscid bile was aspirated from the gall bladder. It was apparent that the mechanical kink of the cystic duct had interfered with the emptying of the gall bladder, producing stasis, infection, and stone formation. In spite of the fact that the common duct appeared perfectly normal, we decided to explore it. A longitudinal incision permitted the escape of dark brown, tenacious bile laden with flakes of biliary pigments. Irrigation with physiological saline solution dislodged a considerable amount of inspissated bile pigments and mucus. Exploration with scoops and probes demonstrated a pseudo-obstruction at the intestinal orifice of the common duct. When, however, stronger pressure was exerted, the instruments suddenly slipped into the duodenum, indicating that the mechanical impediment had been overcome. It then occurred to us that this pseudo-obstruction might be due to a spasm of the common duct sphincter. A cholecystectomy was performed without any difficulty.

Twenty-four hours following operation, 40 cubic centimeters of lipoiodine (Ciba) was injected into the drainage tube and permitted to flow into the common duct. Roentgenographic studies disclosed a pronounced dilatation of the entire biliary tree. The common duct measured 14 millimeters in diameter which was 2½ times its normal size, while the right and left hepatic ducts were about 1½ times larger than usual. The walls of the dilated ducts were smooth and regular in contour, showing no evidence of stones, strictures, or neoplasms. The remnant of the old cystic duct was dilated so that it formed a small sacculation. Apparently there was some impediment at the ampulla for the lipoiodine was confined to the bile ducts, none having escaped into the duodenum (Fig. 1).

A second roentgenogram taken 25 minutes later revealed the lipoiodine to be retained in the bile ducts. After three hours, the radiopaque oil was seen to trickle into the duodenum. Evidently the temporary obstructive agent or spasm had momentarily disappeared, permitting the dilated bile ducts to eject their oily content into the intestinal tract. Skiagrams taken 48 hours after the primary injection exhibited a slight retention of lipiodol in the hepatic and common ducts.

It is to be remembered that before exploring the common duct it appeared to be fairly normal; however, the visualization studies demonstrated a distinct dilatation of the entire biliary tract. Perhaps such a distention is much more common than is customarily believed. The operative exploration of the choledochus disclosed what was thought to be a spasm and hypertrophy of the sphincteric mechanism. Roentgenograms confirmed this impression by showing that there was sufficient spasm of the sphincter to cause a retention of lipiodol and it required 3 hours' time for the sphincterismus to subside sufficiently to permit the oil to enter the duodenum. If this obstruction were caused by an inflammatory reaction of the ampulla and duodenal wall, as suggested by Strauss, then it should have persisted, exhibiting no periods of partial relaxation during which the lipiodol could escape into the bowel. Thus it would seem that the spastic sphincteric mechanism acts as a mechanical hindrance to the physiological flow of bile, producing an increased intraductal pressure with subsequent dilatation of the biliary radicals and ducts, causing a cholangiectasia and even hydrohepatosis.

Does the choledochal sphincter have sufficient contractile power to overcome the normal intrahepatic and intraductal secretory

Fig. 2, left. Case 2. Roentgenograms taken 5 minutes after injecting lipoiodine in common duct. Note the marked dilatation of the large bile ducts. The oil had not entered the duodenum because of a choledochal obstruction near the ampulla. Was this a sphincterismus?

Fig. 3. Case 2. Plate taken 30 minutes later showed the lipoiodine to be in the intestinal tract. There was still a slight stasis of oil in the common duct. The obstructing mechanism (Fig. 2) had completely disappeared, which suggested a relaxation of the choledochal sphincter.

pressure and produce a chronic retention of the biliary products? Whitaker has demonstrated that bile enters the gall bladder by flowing up the cystic duct, but it does this only when the sphincter of Oddi is closed. The existence of this sphincter as an anatomical and physiological structure has been much discussed, and it is now generally accepted as being a functioning unit. Its physiological action has been demonstrated by Lueth, who by means of an ingenious apparatus measured the pressure gradients in the intramural part of the common biie duct and obtained positive evidence of its sphincteric action. He showed that the ampulla could be distended with perfusion fluid, indicating that the terminal part of the bile duct must be able to retain fluids under positive pressure. This confirmed the previous findings of McMaster and Elman who had measured the pressure in different parts of the extrahepatic biliary system by means of triple intubation. Judd and Mann, and later Lueth, proved that the sphincter action was not produced by the tone of the duodenal wall. Cole, and Elman and McMaster found that, in a dog, the sphincter could hold up bile in the common duct at a pressure of 100 millimeters of water, while Judd and Mann found the ampulla able to withstand a pressure of 600 to 675 millimeters of water. Whitaker cut the sphincter of Oddi and found that the pressure within the common bile duct fell to zero and that the gall bladder did not fill. Ivy and Sandblom believe that motor dysfunctions of the sphincter of Oddi are capable of producing bile stasis and simulate the syndrome cholecystitis and biliary colic.

Such physiological evidence confirms our observation that a spasm of the choledochal sphincter is capable of producing and does produce bile retention by interfering with the normal emptying of the extrahepatic ducts.

CASE 2. Mrs. L. L., housewife, aged 32 years, complained of recurrent attacks of severe biliary colic for the past 3 years. About 4 months prior to admission, a cholecystectomy was performed and the gall bladder was found to be filled with calculi. Two weeks after the operation, the colic-like pains associated with nausea, vomiting, and jaundice, recurred. Since then she has had several acute paroxysms.

She was a nervous, emaciated woman. No tenderness could be elicited in the right subcostal area. The liver was not enlarged and the kidneys were not palpable. Roentgenograms of the abdomen and retrograde pyelograms depicted a round stone lying at the level of the second lumbar vertebra external to the urinary system; hence it was thought to be in the common duct.

Fig. 4, left. Case 3. On the seventh postoperative day, lipoiodine was introduced into the cholecystostomy tube; only a very small amount of the oil passed into the common duct for the cystic duct was practically occluded.

Fig. 5. Case 3. Six days later a similar injection was made. The lipoiodine outlined the gall bladder, cystic duct, and dilated bile ducts. There was a mechanical blockade at the lower end of the choledochus which caused a retention of oil. Radiographic studies made 30 minutes later revealed a relaxed sphincter of Oddi for the oil was flowing into the duodenum.

Upon opening the abdominal cavity, the liver capsule was found to be striated with white fibrous tissue. There was pronounced dilatation and enlargement of the common duct caused by a calculous obstruction in its retroduodenal portion. As soon as the choledochus was incised, clear dark brown bile escaped, exposing a large, mushy, granular stone which was removed by a scoop. Instrumental and digital examination indicated that the common duct was patent. A definite impediment, however, was encountered at the intestinal orifice of the duct and moderately sustained pressure was required before the resisting force was suddenly overcome and the curved hemostat entered the duodenum. A No. 22 soft rubber catheter was anchored into the common duct for drainage.

Twenty-four hours following operation, 45 cubic centimeters of lipoiodine (Ciba) was injected into the common duct. The radiopaque oil demonstrated a pronounced dilatation of the bile radicals. In fact, the persistent intraductal pressure had been so great that it had caused the stump of the cystic duct to form a small diverticulum. No organic lesion could be demonstrated but the lipoiodine failed to flow into the duodenum (Fig. 2). Thirty minutes later the oil was seen to be in the upper jejunum (Fig. 3). What was this evanescent obstruction in the region of the ampulla of Vater? Tumors, strictures, neoplasms, or inflammatory reactions should cause a permanent blockade, but to find that the apparent obstacle had disappeared within 30 minutes' time, permitting a complete evacuation of the biliary tree, certainly would suggest a functional derangement of the sphincter of Oddi.

One naturally wonders whether there is a causal relationship between the motor dysfunctions of the choledochal sphincter and the presence of stones and infection. It is conceivable that an inflammation of the ampulla and the duodenum might occasionally increase the hypersensitivity of the sphincter and induce a spasm; on the other hand, a primary sphincterismus could easily cause a retention of bile, and stagnant bile on becoming infected would favor the formation of stones. It was impossible to determine whether the stone or the spasm of the sphincter was the provocative agent in this particular case. Undoubtedly most of the patient's symptoms were caused by the stone in the common duct but after the calculus had been removed the bile stasis still persisted because the abnormal physiological tonus of the choledochal sphincter prevented the bile from entering the intestinal tract. Our experience with 7 other cases clearly demonstrates that it is possible for the common duct sphincter to function normally in the presence of generalized inflammation of the biliary tract associated with stones, strictures, and even a pancreatitis. This conception agrees with the observation of Saralegui who injected lipiodol into persisting external biliary fistuls of cholecystecto-

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 6. Case 4. On the fifth postoperative day, 40 cubic centimeters of lipoiodine was introduced into the common duct. Note the extensive dilatation of the bile ducts. Roentgenograms taken 3 minutes after the injection of the oil demonstrated a blockade at the lower end of the choledochus.

Fig. 7. Case 4. Second roentgenogram taken 10 minutes later. The obstruction was beginning to disappear as the oil was now slowly trickling into the duodenum.

Fig. 8. Case 4. Studies made on the ninth postoperative day, 35 minutes after the original injection. The common duct was beginning to empty more rapidly. It required 1 hour's time for all the oil to enter the duodenum and yet no obstructive agent could be demonstrated at the operating table. An evanescent obstruction that completely occludes the common bile duct and then suddenly disappears seems to be suggestive of a choledochal sphincterismus.

mized individuals. He encountered considerable difficulty in visualizing the biliary tract because the radiopaque oil entered the common duct and immediately passed through the relaxed sphincter of Oddi into the duodenal lumen. Infection, then, does not necessarily cause a sphincterismus.

Case 3. Two weeks ago, Mrs. V. C., housewife, aged 43 years, had her first attack of "gall-stone trouble." It was characterized by a severe colic-like pain most intense in the right subcostal area and radiating to the interscapular region. One week later, a firm tender mass could be felt in the right upper quadrant. The liver and spleen were not palpable but the abdominal muscles were slightly spastic. Her skin and scleræ were deeply jaundiced, the icteric index being 88. Roentgenograms demonstrated a non-functioning gall bladder without evidence of stone formation.

Upon exposure of the gall bladder, it was found to be about three times normal size and to contain several stones. Its walls were acutely inflamed, thickened, and indurated. The serosa was studded by small petechial hemorrhages. The common duct was dilated and its fibrosed walls were firmly adherent to the duodenum and the fundus of the gall bladder. A large stone blocked the choledochal lumen. Sixty cubic centimeters of a thick, yellow, purulent bile was aspirated from the gall bladder, thus confirming the diagnosis of acute cholecystitis. The calculi were removed from the gall bladder, and the cystic duct was found to be completely occluded by the inflammatory swelling and edema. The dilated common duct was opened and the single calculus removed. The pancreas and ampulla of Vater were normal. Scoops, probes, and irrigating

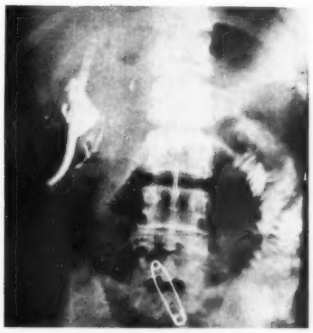

Fig. 9. Case 5. Eight days following removal of an acutely inflamed gall bladder, 40 cubic centimeters of lipoiodine was injected into the common duct via the cystic duct. There was no evidence of bile stasis or dilatation of the ducts. The choledochal sphincter was relaxed for the oil immediately entered the duodenum.

Fig. 10.

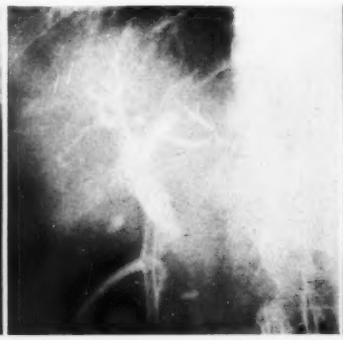

Fig. 11.

Fig. 12.

Fig. 10. Case 6. Visualization studies on the twelfth postoperative day disclosed a relaxed choledochal sphincter for the oil passed unhindered into the duodenum. The common duct was dilated. The finer biliary radicals did not visualize because a coexisting cholangitis had plugged the smaller ducts and because the relaxed sphincter permitted the oil to escape.

Fig. 11. Case 6. On the twenty-ninth postoperative day the finer bile ducts were visible because the prolonged catheter drainage had reduced the inflammatory reaction in the ducts. The distal segment of the common duct did not visualize because the sphincter of Oddi had relaxed and the lipoiodine was in the duodenum.

Fig. 12. Case 6. Showing the marked reduction in the size of the biliary duct following 33 days of continuous drainage.

fluids entered the duodenum without encountering any hindrance, therefore proving the common duct to be patent. Because of the chronic stasis of bile and the acute infection of the biliary tract, both the gall bladder and the common duct were drained.

Seven days following the operation, 40 cubic centimeters of lipoiodine (Ciba) was introduced into the gall bladder through the cholecystostomy tube. The oil escaped around the drainage tube and only a very small amount entered the common duct, which confirmed the operative findings of an inflammatory occlusion of the cystic duct (Fig. 4). Six days later a second roentgenogram was made following the instillation of 60 cubic centimeters of lipoiodine. The gall bladder was easily filled and the oil immediately passed into the choledochus because the cystic duct was now open. During the introduction of the radiopaque oil, the patient complained of severe epigastric pain similar to her previous colic-like attacks. Evidently the common duct was being distended but what was preventing the oil from entering the duodenum? Roentgenographs taken immediately after the injection of the oil indicated that there was a mechanical blockade at the intestinal orifice of the common duct causing a retention of the lipoiodine (Fig. 5). X-ray films taken 30 minutes later disclosed that this temporary obstructing mechanism had completely disappeared for the lipoiodine was seen to be in the duodenum and upper jejunum.

This study presents roentgenographic evidence of the futility of attempting immediate drainage of the entire biliary tract by performing a cholecystostomy in cases of acute cholecystitis and cholangitis in which the inflammatory edema has occluded the cystic duct. If such patients are so unfortunate as to have a concomitant spasticity of the common duct sphincter with bile stasis, then the drainage of the gall bladder merely relieves the pressure on that organ without mitigating the stasis within the ductal system. It was interesting to note that the patient complained of the typical pains of biliary colic during the interval when the spastic sphincter caused a retention of the lipoiodine and that the pains did not subside until the sphincter relaxed and permitted the oil to enter the duodenum. This again demonstrates that a hypertonicity of the sphincteric mechanism of the common duct can occur in the presence of an acute infection.

Case 4. Mrs. A. C., housewife, aged 50 years, entered the hospital complaining of "gall trouble." She had suffered from paroxysms of nausea and vomiting associated with epigastric distress for the last year and a half. During the past 9 months she had been persistently jaundiced, her stools had been acholic, and she had lost 65 pounds of weight.

Examination showed a mal-nourished, asthenic, deeply jaundiced woman. The liver extended 2 inches below the costal margin. The gall bladder could not be palpated nor did it concentrate the cholecystographic dye. The van den Bergh test was strongly positive in both the direct and indirect

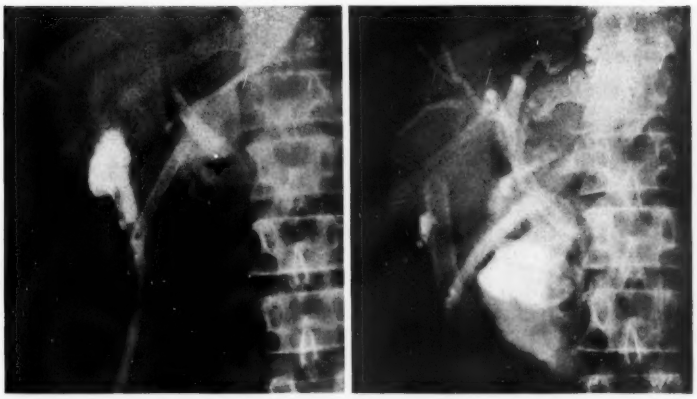

Fig. 13, left. Case 7. Lipoiodine was introduced into the cholecystostomy tube 2 days following operation. The gall bladder was definitely outlined but the oil did not enter the choledochus because of an inflammatory occlusion of the cystic duct. Some of the lipoiodine had escaped around the drainage tubes.

Fig. 14. Case 7. Twenty minutes later lipoiodine was injected into the "T" tube and revealed a dilated common duct. The choledochal sphincter was relaxed, for the oil was seen to be passing into the intestinal tract.

phases. The icteric index was 73. The general appearance of the patient, the rapid loss of weight, and the persistent, but painless, jaundice suggested malignant changes in the hepatobiliary system. After 10 days of preparation, a laparotomy was performed.

The liver was found to be definitely enlarged and its capsule was tense, thickened and scarred. It had a dark green, nutmeg appearance, indicating a chronic retention of bile products. The fundus of the gall bladder was firmly adherent to the common duct with a fistulous communication between the two. Several stones were felt in the gall-bladder cavity. The dilated common duct was opened. On digital examination no calculi were palpated but gentle irrigation with saline solution flushed out some fine gravel, several very small stones, and some inspissated plugs of bile stained mucus. Catheters, probes, and fluids could be forced into the duodenum, thus attesting to the patency of the common duct. The dense adhesions had produced such anatomical distortions that it was technically impossible to perform a complete cholecystectomy. The anterior and lateral portions of the gall bladder were therefore excised, leaving the posterior wall intact in its hepatic fossa. The mucosa of the remaining gall bladder tissue was destroyed by carbolization and the muscular coats firmly approximated by sutures. A No. 22 catheter was placed in the hepatic duct and a No. 20 catheter into the common duct for drainage purposes.

On the fifth postoperative day, 40 cubic centimeters of lipoiodine (Ciba) was introduced into the common duct. X-ray photographs taken 5 minutes later verified the extreme dilatation of the bile ducts, particularly of the right and left hepatic ducts. Some of the lipoiodine had escaped along the drainage tube and had outlined the old gall bladder fossa. At the duodenal orifice of the common duct the lipoiodine had encountered an obstacle (Fig. 6) and even 10 minutes after injection only a few drops of oil had entered the duodenum (Fig. 7). Due to the general condition of the patient, we felt it inadvisable to prolong the fluoroscopic studies. Four days later 60 cubic centimeters of lipoiodine were instilled into the common duct, again demonstrating an obstruction in the region of the ampulla. Roentgenographic studies were made 35 minutes later and the lipoiodine was seen to be flowing past the sphincter of Oddi into the intestinal tract (Fig. 8). Evidently the sphincter had relaxed and the oil had free passage to the gastro-intestinal canal.

COMMENT

These 4 cases of chronic bile stasis which have been presented as examples of choledochal-sphincteric dysfunction were all associated with calculi or infection of the bile ducts. Are we justified in assuming that there is no causal relation between the functional disturbance of the sphincteric mechanism and the co-existing stones and infections? Does the inflammatory reaction increase the hypersensitivity of the choledochal-duodenal-sphincteric system, resulting in a spastic contraction or sphincterismus? Might not the mal-functioning, hypertonic sphincter

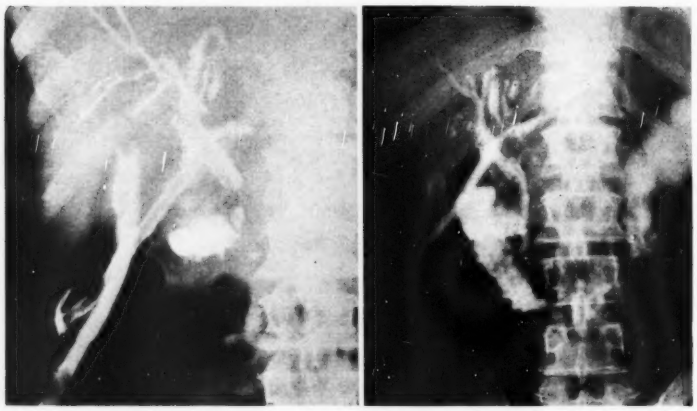

Fig. 15, left. Case 7. On the eighteenth postoperative day, 60 cubic centimeters of lipoiodine was injected into the cholecystostomy tube. The cystic duct was then patent for the oil entered the choledochus and duodenum. This is in contrast to Figure 13 which shows the occluded cystic duct.

Fig. 16. Case 7. Roentgenogram made after the bile ducts had been drained for 18 days. Note the marked reduction in their size. The choledochal sphincter is relaxed. (Compare with Fig. 14 as both plates were made following injection of common duct.)

cause a retention of bile, the stagnant biliary secretions then become infected and favor the formation of calculi? In order to answer these questions the following studies were carried out.

CASE 5. Mrs. L. B., housewife, 41 years of age, had had severe "gall-stone attacks" for the past 2 years. The acute exacerbations were frequently associated with nausea, vomiting, and prostration, but no jaundice.

Fig. 17. Case 8. Lipoiodine was injected into the cystic duct. The bile ducts were not visualized even though the roentgenograms were made immediately after the introduction of the oil. The relaxed choledochal sphincter had permitted the oil to enter the duodenum.

Physical examination disclosed a tenderness in the right hypochondrium but the gall bladder was not palpable. The van den Bergh reaction did not evidence a bile retention and the icteric index was 4.5. Radiological studies depicted a non-functioning gall bladder filled with small calculi.

At operation the liver was found to extend 1½ inches below the right costal margin. Its capsule was thickened and scarred, particularly in the region of the gall-bladder fossa. The walls of the gall bladder were inflamed, hyperemic, and edematous. Dense adhesions held it in firm apposition to the duodenum. The inflammation and edema had obliterated the lumen of the cystic duct. The choledochus appeared normal except for a slight fibrosis of its walls, there being no evidence of stone, stricture, dilatation, or neoplasm. In fact, there was nothing to arouse suspicion or to warrant drainage of the common duct. The gall bladder was removed and for therapeutic as well as investigative reasons a No. 16 catheter was inserted through the stump of the cystic duct to drain the common hepatic radicals.

On the eighth postoperative day, 40 cubic centimeters of lipoiodine (Ciba) was injected into the drainage tube. Roentgenograms made immediately after this injection failed to show any evidence of bile stasis or dilatation of the bile ducts; in fact, the major portion of the oil had already entered the jejunum, indicating a relaxed choledochal sphincter (Fig. 9).

This case represents a generalized infection of the entire biliary tree. The diffuse scarring, fibrosis, and lymphocytic infiltration of the

cystic duct and gall bladder indicate a chronic inflammatory reaction. The extrahepatic bile ducts were not dilated but the infection had produced a fibrosis and induration of their walls. In spite of the long standing infection of the biliary ductal system, however, the choledochal sphincter functioned normally, permitting the lipoiodine to enter the duodenum immediately. Thus, it would seem that infection *per se* does not always initiate a functional derangement of the sphincteric mechanism of the ampulla. It also demonstrates that a chronic infection of the hepatic system does not cause a dilatation of the bile ducts unless there is a co-existing obstruction, such as stones, strictures, kinks, tumors, or sphincterismus.

CASE 6. C. M., aged 69 years, had had "stomach trouble" for 35 years, characterized by bloating, belching of gas, indigestion, and passage of flatus. Over a period of 8 years he had suffered from recurrent attacks of jaundice, at which times his stools were acholic and his urine acquired a very dark color. He had lost 55 pounds of weight during the past 10 months.

Examination disclosed the skin to be deeply jaundiced. There was a large, firmly fixed mass in the epigastric area. The liver, spleen, gall bladder, and kidneys were not palpable. Cholecystograms revealed a non-functioning gall bladder with several calculi apparently in the common duct. During his pre-operative period he had several chills with a febrile reaction of 106 degrees F., which suggested a cholangitis secondary to an intermittent calculous obstruction. The large tumor mass, the rapid loss of weight, and the persistent jaundice made the diagnosis of pancreatic malignancy associated with cholelithiasis seem very plausible.

A laparotomy exposed a dark green, nutmeg liver, somewhat enlarged, with a firm, tense fibrotic capsule. The gall bladder was about twice normal size, and its walls were very thick, indurated, and edematous. The cystic duct was patent. The common duct was about three times greater than normal. After aspirating 60 cubic centimeters of black viscid bile, several small stones could be felt in the hepatic ducts. The pancreas was somewhat enlarged, firm, and indurated, but did not present the stony hardness of a malignancy. A cholecystectomy and choledochostomy were performed after removal of several small stones from the hepatic ducts. Painstaking examination failed to show any gross obstruction at the ampulla of Vater.

Visualization studies were deferred until the twelfth postoperative day, at which time 40 cubic centimeters of lipoiodine (Ciba) was injected into the choledochus. Roentgenograms were taken immediately and evidenced a marked dilatation of the common duct. The hepatic ducts were not outlined because the relaxed sphincter had permitted the oil to escape into the duodenum (Fig. 10). These same findings were present on similar radiographic studies made on the twenty-ninth (Fig. 11) and thirty-third (Fig. 12) postoperative days. They corroborate Whitaker's assertion that the gall bladder and bile ducts do not become filled or distended if the sphincter of Oddi is relaxed.

These observations signify that a chronic biliary stasis, hepatic congestion, dilatation of the bile ducts, the presence of calculi in the ducts, and a generalized infection of the biliary system, with a concomitant pancreatitis, apparently do not always increase the tonicity of the sphincter of Oddi or interfere with its ability to relax.

CASE 7. J. H., farmer, 53 years of age, entered the hospital because of a "gaseous indigestion." During the past 4 years he had been annoyed by the passage of flatus, belching of gas, and epigastric distress. Intermittent attacks of sharp colic-like pains in the right subcostal area frequently required morphine for their relief. He denied having been jaundiced.

Just beneath the right costal margin there was a firm, tender, ovoid mass. The liver did not seem to be enlarged. The skin and sclera were definitely jaundiced, the icteric index being 12.

Exploration revealed a moderately distended gall bladder which contained many small calculi. The cystic duct contained no stones. The common duct was twice its regular size and, in its retroduodenal portion, there was a small lobulated mass which was thought to be either an enlarged lymph node or an incarcerated intramural calculus. Upon opening the common duct a well developed annular stricture was found just distal to the junction of the cystic and hepatic ducts, which had diminished the diameter of the choledochus to about one-fourth its original size. Technically it was impossible to excise the stricture and then establish the continuity of the bile ducts, nor could a hepatico-enterostomy be done. A cholecystostomy and choledochostomy were performed as these conservative procedures would permit a cholecysto-enterostomy to be done at a later date should the inflammatory scar tissue occlude the common duct. Careful examination disclosed that the intestinal portion of the common duct was patent; therefore, the fibrous band was divided and a "T" tube inserted in such a manner that one limb of the "T" dilated the constricted portion of the duct.

Two days after operation 40 cubic centimeters of lipoiodine (Ciba) was introduced into the gall bladder through the drainage tube. Roentgenograms were taken immediately following the injection and again in 20 minutes. In both instances the gall bladder was clearly outlined but the radiopaque media had been unable to traverse the occluded

cystic duct (Fig. 13). This again shows that a cholecystostomy does not always decompress the liver and biliary ducts. Lipoiodine was then injected into the "T" tube, thus outlining the dilated common duct. The relaxed sphincter of Oddi permitted the major portion of the oil to escape into the duodenum (Fig. 14).

Sixteen days later, 60 cubic centimeters of lipoiodine was injected into the gall bladder and much to our surprise we found the cystic duct to be patent (Fig. 15). This demonstrated the value of prolonged drainage in cases of biliary stasis, for decompression of the gall bladder and common bile duct had permitted the inflammatory edema of the cystic duct and biliary radicals to subside (Fig. 16). The hepatic ducts and the proximal three-fourths of the common duct were of normal size and contour, but the distal one-fourth, the portion beyond the lower limb of the "T" tube, could not be visualized. Was there an obstruction in the lower end of the "T" tube which prevented the oil from entering the ampulla of Vater? Apparently not, for the lipoiodine was seen in the upper jejunum. The relaxed sphincter of Oddi had permitted the lower segment of the common duct to evacuate its oily content into the intestinal lumen.

Here was confirmatory evidence that gall stones, cholecystitis, inflammation, strictures, fibrosis of the walls of the bile ducts, and even the presence of a foreign body such as the "T" tube, do not necessarily interfere with normal relaxation or function of the choledochal sphincter.

CASE 8. Mrs. A. A., housewife, 29 years of age, had had gall-bladder colic since her first pregnancy two and one-half years ago. Frequent exacerbations followed the initial attack, associated with nausea, vomiting, jaundice, and acholic stools. Physical examination and laboratory studies were normal with the exception of two cholecystograms which demonstrated a poorly functioning, dilated gall bladder containing several stones.

When the abdomen was entered, the gall bladder was found to contain three stones, and it was distended by a yellow brown bile. The walls of the cystic duct were indurated, edematous, and fibrosed. The common and hepatic bile ducts were normal, but the head of the pancreas was somewhat enlarged and indurated. After performing a cholecystectomy a No. 22 catheter was inserted into the stump of the cystic duct to permit visualization studies.

Forty cubic centimeters of lipoiodine was introduced into the common duct. The bile ducts were not visualized because the oil immediately entered the duodenal tract. An additional 40 cubic centimeters of lipoiodine was injected through the drainage tube, but it flowed unhindered into the intestinal lumen; hence, the sphincter of the common duct was relaxed (Fig. 17).

CASE 9. Mrs. A. A., housewife, 41 years of age, entered the hospital with the diagnosis of "gall stones." During the past 9 months she had had several attacks of biliary colic. Examination and laboratory studies failed to show any abnormalities except a non-functioning gall bladder.

At operation the liver was found to be of normal size; its capsule was scarred, thickened, and fibrosed. The gall bladder was moderately distended and its walls were thick, indurated, and impregnated with fat. Two large stones were incarcerated in a saccular dilatation at the junction of the gall bladder with the cystic duct. A viscid, bile stained secretion was aspirated from the gall bladder, from which luxuriant cultures of Bacillus coli and gram negative bacilli were obtained. The common and hepatic ducts were not dilated, nor could stones be palpated; their walls were slightly indurated. The ampulla appeared to be normal. A cholecystectomy was performed and a No. 20 catheter sutured into the cystic duct for functional studies.

Forty-eight hours later 40 cubic centimeters of lipoiodine were injected into the choledochus through the drainage tube. The bile ducts were not visualized for the oil had escaped into the duodenum. This roentgenogram is similar to that in Case 8 and is therefore not presented.

ANALYSIS OF STUDIES

Analysis of these studies indicates that some individuals may have a dysfunction of the choledochal sphincter resulting in a chronic retention of bile, while others may have a normally functioning sphincteric mechanism even in the presence of infection, stones, strictures, kinks, and associated pancreatitis. Such a concept is compatible with the observation that all sphincters of the body may at some time or other deviate from the normal by becoming hypertonic or atonic. A spasm of the sphincteric mechanism of the cardiac end of the stomach results in the stasis of food in the esophagus followed by regurgitation, in spite of the increased peristaltic action which attempts to overcome the physiological obstruction. A hypertonicity of the pyloric sphincter often initiates retention of gastric content; a spasticity of the anal sphincters may cause constipation, and the hypertonic contraction of the vesical sphincter often interferes with the complete emptying of the urinary bladder. Then why should we not expect a dyssynergia of the choledochal sphincter?

Even though the spastic bile duct sphincter produces an anatomic blockade of the chole-

dochus, it may still respond to the proper physiological and pharmacological stimuli. While studying the effects of secretin-cholecystokinin injections on the duodenal secretions of normal subjects, Ivy and his collaborators made some interesting observations. In one instance, they passed the duodenal tube, and at first recovered normal bile, but all at once the flow of bile stopped. This they attributed to a spasm of the choledochal sphincter. Then a solution of secretin-cholecystokinin was injected. A copious flow of pancreatic juice resulted which was not bile stained. Ten minutes later, the patient complained of distress in the right subcostal area. Within 50 minutes, the pain was so severe that relief was sought. Magnesium sulphate was then introduced into the duodenum and a sudden gush of bile evidenced a relaxation of the common duct sphincter. The pain disappeared with the diminution of the intraductal pressure.

Westphal demonstrated that a spasm of the common duct sphincter is capable of producing biliary colic. He inserted a tube into the duodenum and initiated evacuation of the gall bladder by introducing olive oil. After aspirating several samples of duodenal bile, which signified the sphincter of Oddi was relaxed, he gave a rather large dose of pilocarpine. (In dogs, this drug causes the gall bladder to contract, but prevents the evacuation of the common duct by causing a contraction of the choledochal sphincter.) After administering the pilocarpine, the flow of bile into the duodenum stopped and the patient complained of gall-bladder distress. This pain was not relieved until sufficient atropine was given to overcome the effects of the pilocarpine, at which time bile could again be recovered from the duodenum. Such experiences indicate that the choledochal-sphincteric mechanism can contract with sufficient force to prevent the bile from entering the duodenum and that the resultant increase in intraductal pressure can produce pain.

Biliary dyssynergia offers a plausible explanation for several misunderstood clinical problems.

First, it presents a rational physiological conception of the so called "gall-stone colic of Krukenberg" and "hepatic neuralgia" in which one finds a distended but normal appearing gall bladder without evidence of gross obstruction. A spastic common duct sphincter interfering with the free egress of bile into the duodenum produces a sufficient back pressure to dilate the bile ducts and gall bladder, thus causing pain and discomfort. The absence of infection would account for the normal appearance of the gall-bladder wall. Such patients are not always relieved by a cholecystectomy as this apparently does not correct the dysfunction of the bile-duct sphincter. Possibly the removal of an appendix which was causing a reflex spasm of the sphincter, or the correction of bowel habits, or dietary regulation, may account for improvement in some cases.

Second, a dyssynergia of the choledochal-sphincteric mechanism presents a rational concept for the occurrence of cholangitis and inflammation of the biliary tree, particularly those varieties not preceded by stones or strictures. The hypertonic sphincter favors a retention of the biliary secretions, the stagnant bile then becomes infected, and the infection disturbs the secretory and absorptive functions of the gall bladder and ducts as well as upsetting the delicately poised chemical equilibrium of the bile components, thus initiating the formation of calculi. In such cases the removal of the diseased gall bladder may not be entirely curative, for it does not necessarily change the intrinsic motor derangement of the common duct sphincter.

Strauss operated on 22 patients having a generalized infection and dilatation of the extrahepatic biliary system in which he was unable to demonstrate any obstructive agent. He felt that the bile stasis was secondary to an inflammatory duodenitis thus confirming the observations of Crain and Walsh that an experimentally induced duodenitis interferes with evacuation of the gall bladder. While it is possible that an inflammatory swelling of the duodenal wall could occlude the common duct, such instances must be extremely rare for in 7 patients having a generalized infection of the biliary tract, we failed to find any evidence of obstructing duodenitis. In 5 instances, the common duct sphincter was com-

pletely relaxed and the lipoiodine passed unhindered into the intestinal canal. In the 4 patients having an apparent obstruction at the duodenal end of the common duct, no evidence of a duodenitis was found. The ducts were patent and the blockade was evanescent. An inflammatory obstacle should not disappear in 10 minutes or 35 minutes after the introduction of the oil. Lipoiodine visualization of the common bile duct, supplemented by direct operative examination indicates that bile retention is caused by a spasticity of the sphincter of Oddi and not by a duodenitis.

Third, a dyssynergia of the choledochal sphincter could be responsible for the persistent gall-bladder distress which is occasionally not relieved by a cholecystectomy. The spastic dysfunctioning sphincter inhibits the free escape of the liver bile into the duodenum, causing an accumulation of bile and increasing the intraductal pressure until the extrahepatic radicals become dilated. Judd and Mann have demonstrated that the dilated bile ducts may contain more bile than a normal gall bladder. The dilatation usually continues until the sphincter itself becomes dilated or is unable to withstand the increased pressure. If the sphincter of Oddi is destroyed, no such dilatation occurs. Following removal of the gall bladder, Puestow noted that the bile did not emerge into the duodenum by spurts as it does normally, but that there was a constant dribble, indicating an overflow retention. Repeated lipoiodine visualization studies were made on 4 cholecystectomized patients for as long as 33 days after their operations, and each of them exhibited a contracted, hypertonic sphincter which was producing a physiological block of the common duct. In one instance the sphincterismus had produced such an increased intraductal pressure that the stump of the cystic duct became dilated, forming a diverticulum. These dilatations were not due to a neurogenic atonia of the bile ducts attributed to the operative intervention, because roentgenographic studies evidenced an increased tone—even a hyperspasticity of the sphincter. Five other cholecystectomized individuals had an atonic, relaxed sphincter when similarly examined. Apparently then this sphincteric mechanism does not always become paralyzed following the extirpation of the gall bladder, but in some cases its persistent spasm may continue to produce a physiological block resulting in pain and distress.

Relief of these persistent symptoms depends on obtaining a relaxation of this spastic sphincter. Westphal has demonstrated that it is innervated by the autonomic nervous system and hence the hypertonicity can be relieved by atropine. Meltzer, Lyons, and Ivy agree that the intraduodenal instillation of magnesium sulphate immediately relaxes the choledochal sphincter and permits the evacuation of the bile ducts. Newman accomplishes the same end by using olive oil. Such operative measures as a choledochal enterostomy or a longitudinal division of the sphincter of Oddi could possibly be employed to overcome this increased intraductal pressure; however, such radical procedures should not be advocated until we have a more accurate physiological concept of biliary dyssynergia. It seems that medical regimen will do much to minimize the discomfort caused by a spasticity of the choledochal sphincter.

TECHNIQUE

A uniform technique was followed throughout these studies. Two parts of lipoiodine (Ciba) and one part of olive oil were heated to body temperature and the resulting mixture was permitted to flow into the drainage tube by gravity. Positive pressure was used in only those cases having a spastic contraction of the choledochal sphincter. In such instances the patients complained of hepatic distress before sufficient pressure could be used to overcome the hypertonic sphincter. It was found that if the patient was placed in the Trendelenburg position during the introduction of oil the biliary radicals were better visualized. Roentgenograms were taken immediately following the introduction of the oil. If there was a retention of lipoiodine in the bile ducts then subsequent roentgenograms were taken at 25 and 35 minute periods. Fluoroscopic studies were made in some cases.

One naturally wonders whether the lipoiodine itself might not cause a contraction of the sphincter and a retention of the oil but

the demonstration of a relaxed sphincter in 5 cases would seem to negate such a criticism. Oils, particularly olive oil, are active cholagogues; therefore, they should expedite the evacuation of the biliary tract rather than favor a stasis.

SUMMARY

1. Clinical and experimental investigations indicate that a hypertonicity or dyssynergia of the choledochal sphincter may mechanically interfere with the evacuation of the gall bladder and bile ducts, thus producing a stasis of bile with a dilatation of the biliary radicals. Such a sphincterismus was demonstrated in 4 patients by direct roentgenographic visualization of the bile ducts following the introduction of lipoiodine. In one instance spastic contraction of the common duct sphincter persisted for 3 hours before it relaxed and permitted the lipoiodine to enter the duodenum.

2. The dyssynergia of the common duct sphincter may be independent of, or associated with, a generalized infection of the biliary tract, the presence of stones, strictures, kinks, or pancreatitis. The extirpation of the gall bladder, the removal of the stones, the division of the stricture, and the drainage of the infected bile ducts do not always overcome the spasticity of the sphincter, for, in some cases, it persisted after these operative measures.

3. Physiological evidence indicates that the choledochal-duodenal-sphincteric mechanism has sufficient contractile force to prevent the flow of bile into the duodenum, thereby increasing the intraductal pressure and causing pain and discomfort. Such a concept offers a rational explanation for occurrence of "gallstone colic" in the absence of stones or infection, and for the so called "hepatic neuralgia" and accounts for the persistence of gall-bladder distress in some cholecystectomized patients.

4. A dyssynergia, or spastic dysfunction of the choledochal sphincter, provides an anatomical blockade of the common duct, resulting in a retention of bile. The stagnant bile becomes infected, and calculi are then precipitated. In such cases a cholecystectomy would not necessarily be curative, for following the removal of the gall bladder, the intrinsic spasm of the common bile-duct sphincter may continue. Four patients having a spasticity of this sphincter were studied by lipoiodine visualization for as long as 33 days following a cholecystectomy, and the sphincterismus still persisted.

5. It is very probable that the proper postoperative medical regimen, including those substances which relax the choledochal sphincter such as atropine, magnesium sulphate, or fats, would do much to correct the abnormal spasticity of the choledochal sphincter and thus tend to minimize the unsatisfactory results which sometimes follow cholecystectomies.

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PRACTICAL ROENTGEN PELVIMETRY

A Comparison of Methods in 100 Cases

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EXTERNAL pelvimetry, while valuable, may be fallacious. There are many occasions upon which the obstetrician may be greatly benefited by a more accurate knowledge of the measurements and shape of the female pelvis, and of the relationship between the fetal head and its bony canal. Roentgen pelvimetry is a simple procedure and one may employ it without any fear of damaging the fetus. Whereas it may not be practical or necessary to examine all patients roentgenologically there are certain definite indications. Freiheit states all primiparæ with a diagonal conjugate of 11.5 centimeters or less, or in whom the vertex fails to engage from the thirty-second to the thirty-fourth week, or in whom an unusual presentation occurs, should be subjected to X-ray examination. Multiparæ with histories of previous difficult labors with normally sized infants and multiparæ with a history of fetal death subsequent to operative delivery from below should also be roentgenographed. There are also additional indications for roentgen cephalometry, but we shall confine our attention in this publication to pelvimetry. Recently the value of X-ray examination has also become apparent for women in labor in whom there is some unexplained delay.

Several methods of roentgen pelvimetry have been advanced, and these are chiefly based on either a geometrical or stereoscopic principle. Any line of a given length parallel to a film is to the length of its image produced on the plate as the distance between the target and the line is to the distance between the target and the plate (Fig. 1). This is our definition of the geometrical principle. In applying this method to pelvimetry it is necessary to have the pelvic inlet parallel to the plate. This is accomplished by the semirecumbent position with the upper anterior border of the symphysis pubis and the interspace between the fourth and fifth lumbar vertebræ at equal distances from the table top. Hypher, Roberts, and Ewer and Bowen report the use of this method. The method described by Thoms eliminates the calculations herein described. He employs the principle that any object placed at a known distance above the film will be magnified in the same degree as any other object placed at the same distance, if the target-film distance is kept constant. The object, if it is a line, need not be parallel to the film to give the same degree of magnification as any other equal line, as long as both lines are identically placed. In practice, Thoms introduces the perforated centimeter grid in the same plane previously occupied by the superior strait. Jarcho reports the use of the centimeter grid also in the lateral view. Jacobs likewise reports on this method for lateral pelvimetry. He employs a separate film, however, for the perforated grid. Using the geometrical principle, Walton has prepared a chart which gives the corrected reading for each distance measured on the film (Fig. 2). It is necessary to know the distance from the interspace between the fourth

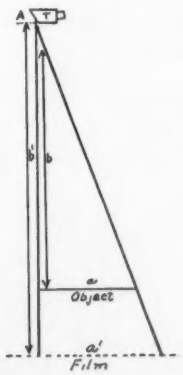

Fig. 1. Geometrical principle involved in determining the actual size of an object placed parallel at a distance from the film. Formula: $a = \frac{a' \times b}{b'}$

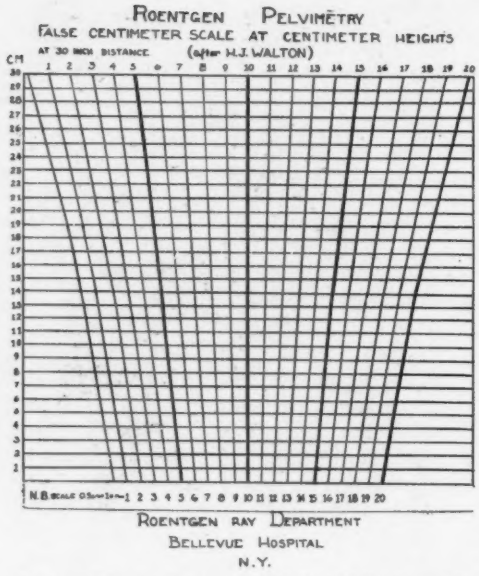

Fig. 2. False centimeter chart (after Walton) used in correcting measurements obtained on semirecumbent and lateral films of pelvis.

and fifth vertebræ to the plate and to have the inlet parallel to the film to apply this chart. Ball and Marchbanks have designed the pelvicephalometer, an instrument which depends on the same principles.

Stereoscopic roentgenometry may be accomplished by reproducing in space the object stereoscoped.

In a method described by Johnson, this is done by superimposition of films on a horizontal view box. Wires are attached to a point above the view box corresponding to the position originally occupied by the target, and the loose ends are placed on the anatomical landmarks of the film between which the measurements are to be made. The divergence of the wires represents the rays. The position formerly occupied by the object being measured is actually reproduced and measured.

Moloy has devised a stereoscopic viewing box equipped with prisms instead of mirrors which projects the image in space. By his method measurements are made of this image while it is being viewed through the prisms. The image is a true optical reproduction of the subject under stereoscopic vision.

COMPARISON OF METHODS

Each method of pelvimetry has certain advantages and certain disadvantages. Stereoscopic technique is superior in its ability to enable one to visualize the relationship between the fetal head and the bony canal. This method also has the added advantage of simplicity in taking the films. The patient may be placed supine, although we have found that better results obtain when the natural lumbar lordosis is increased by placing a sandbag beneath the lumbar vertebræ. The outlet as well as the inlet may be investigated on the stereoscopic plates. However, such methods require additional equipment, often quite expensive. The reading and measuring of the plates in the Moloy stereoscope require constant practice. In the hand of one accustomed to this apparatus very accurate measurements are obtained. However, we have found that the occasional user will have difficulty in obtaining identical readings each time he measures a given pelvis. It must be emphasized though that only stereoscopic methods give the three dimensional sense and, hence, are superior in establishing fetal head canal relations.

Lateral pelvimetry has certain definite advantages. The anteroposterior diameter of the superior strait is usually measurable. The promontory of the sacrum is easily and accurately located. The fetal head is clearly visualized. This view is especially important in demonstrating the curve of the sacrum and the width and shape of the sacrosciatic notch. Jacobs calls attention to the value of determining the inclination of the inlet to the spinal column. As the angle approaches a perpendicular, engagement is facilitated; as it approaches a parallel, engagement becomes more difficult. In taking a lateral view the legs must be extended to obtain this information. From the technical standpoint lateral views are easy to obtain and require no additional equipment. Lateral pelvimetry alone, nevertheless, has one great shortcoming—it does not reveal the shape of the inlet. The transverse or oblique diameters cannot be measured.

The anteroposterior methods of pelvimetry are the ones most in vogue. The semirecumbent position is used, and this may easily be

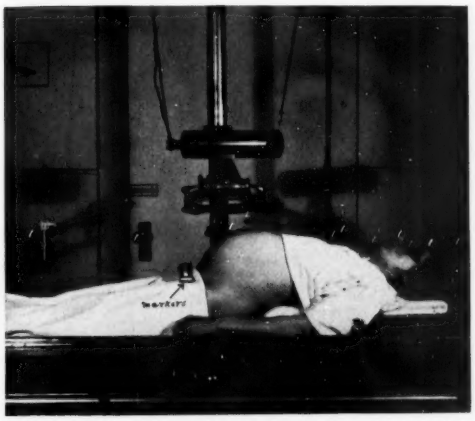

Fig. 3. Patient in position for roentgenography of pelvis for the Moloy stereoscopic study. Note the 9 centimeter markers placed just below the symphysis pubis.

Fig. 4. Roentgenograph obtained (one of stereo set) with patient in position shown in Figure 3. The magnified 9 centimeter distance across markers is shown, which is corrected in the precision stereoscope.

accomplished on a Potter-Bucky diaphragm table with some form of support for the hands and feet. According to the method of anteroposterior pelvimetry selected, certain special, though usually inexpensive equipment is necessary. In the Thoms' method the tube stand must be equipped with plumb lines and a grid must be used for the second exposure. Calipers facilitate the use of this technique. It is not necessary to have the conjugata vera exactly parallel to the cassette if the perforated grid is employed, although the closer the inlet approaches the parallel the better is the demonstration of the shape of the inlet. In the geometrical anteroposterior method and in methods described by Walton with a calculated chart and by Ball and Marchbanks with their instrument and its calculated disc-chart, the plane of the superior straight must be parallel to the film. All these methods do demonstrate the shape of the inlet although at times it is not possible to state definitely exactly what point represents the promontory of the sacrum. The outlet is not accurately measurable by this method although the biischial spinous diameter is obtainable, and it may be considered to be about 3 centimeters above the table top.

TECHNIQUE

In carrying out this study we have used two methods: that of Moloy with the Moloy stereoscope and that of Walton with his chart. In employing Walton's method we have added the lateral view so that we have had an additional check on the conjugata vera measurement.

The stereoscopic films for the Moloy method are obtained with the patient in a recumbent position (Fig. 3). A sandbag is placed under the lumbar spine to alter the pelvic inclination with the plate slightly and thus to facilitate the stereoscopic mensuration. A cardboard strip with two metal markers exactly 9 centimeters apart is placed on the patient just below the symphysis pubis so that the center of the cardboard strip is about at the symphysis and the ends with the metal strips are between the symphysis and the trochanters. The tube is focused over the center of a line joining the anterior superior spines. Target-film distance is 25 inches; stereoscopic shift is 6 centimeters; milliamperes, 30; kilovolt peak, 65; seconds, 12–15. These films are viewed in the Moloy stereoscope although the one employed here does not have the armatures described by him (8). Instead, the prisms are moved back and forth until the 9 centimeter distance between the lead strips on the

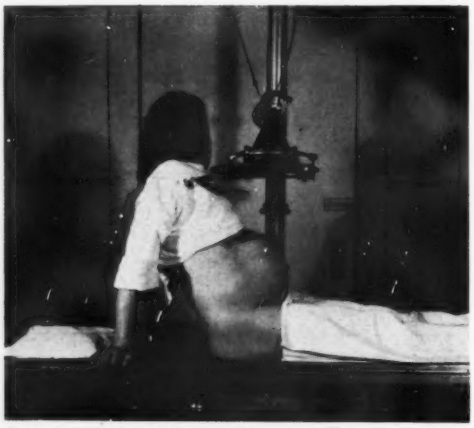

Fig. 5. Patient in position for roentgenography of the pelvic inlet. Measurements adduced here are corrected according to chart in Figure 2.

cardboard marker coincides with 9 centimeters on a ruler held on the opposite side of the prism from the eyes of the observer. Measurements are then made directly on the optical image in space with the ruler. This eliminates the use of a dried pelvis and accomplishes the same thing (Fig. 4).

The anteroposterior view for the Walton method is obtained by placing the patient in a semirecumbent position (Fig. 5). She rests her hands behind her on the side of the table. We have not found any foot support to be necessary although the sandbags may be used here if desired. The interspace between the fourth and fifth vertebræ is marked and its distance from the table measured. The distance from the upper anterior border of the symphysis pubis to the table is also measured. The patient is instructed to arch her back or change her angle of inclination until the two are equal. Then the anteroposterior diameter of the superior strait is parallel to the film (Fig. 6). Exposures are made with the target-film distance 30 inches; milliamperes, 30; kilovolt peak, 72; seconds, 15–20. On the film the distance between the promontory of the sacrum and the posterior border of the symphysis pubis about 1 centimeter below the superior border is measured. This is the magnified conjugata vera. The transverse diameter is measured between the widest points.

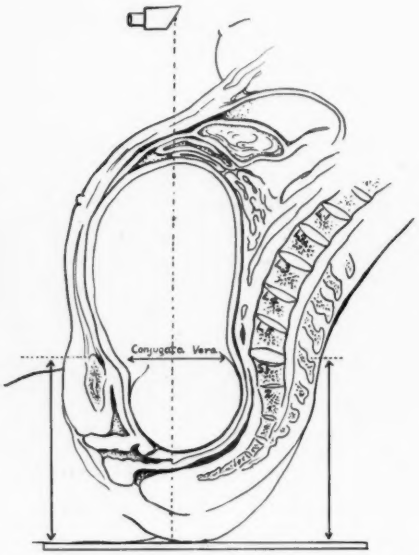

Fig. 6. Schematic diagram of position illustrated in Figure 5 and showing pelvic inlet parallel to film. The distances from the symphysis pubis and fourth lumbar interspace to film are also shown and should be of equal span.

The oblique diameters are measured between the sacro-iliac synchondroses and the opposite ileopectineal eminences, and finally the interspinous diameter is measured (Fig. 7). These measurements are then corrected on the Walton chart. For the correction of the anteroposterior diameter of the superior strait the distance from the interspace between the fourth and fifth vertebræ to the table is added to table top-film distance. Using this correction the true anteroposterior diameter is directly measured off on the Walton chart. In applying this method to the transverse diameter, we have substracted 2 centimeters from the distance measured between the interspace between the fourth and fifth lumbar vertebræ and the table as the transverse diameter of the superior strait is about that distance below the anteroposterior diameter. This was first called to our attention by the publication of Ewer and Bowen and subsequent investigation on dried pelves confirmed the value of such a procedure. For similar reasons we subtract 1 centimeter from the distance between the interspace and table in ob-

Fig. 7. Roentgenograph obtained with patient in position shown in Figures 5 and 6. The magnified anteroposterior and transverse diameters are shown. These are corrected according to the geometrical principle. For expediency the chart of Figure 2 is employed.

taining the oblique diameters, although as Ewer and Bowen point out, the anterior end of the oblique line at the pubic spine is higher than the posterior end at the sacro-iliac synchrondrosis. The distance of the ischial spine from the table top is not measured, but we have considered the biischial diameter to be 3 centimeters from the table top. This is not accurate and indeed in the biischial diameter the stereoscopic anteroposterior film measurements do not check as closely as they do with the other diameters.

The lateral view of the pelvis is obtained by placing the patient on her side with one trochanter directly over the other with her legs extended (Fig. 8). The distance from the midpoint of the interspace between the fourth and fifth lumbar vertebræ to the table is measured. To have a true lateral, the distance between the center of the symphysis pubis and the table should equal this other measurement. The tube is centered over the midpoint of a line connecting the interspace and the symphysis pubis at 30 inches from the plate; milliamperes, 30; kilovolt peak, 72; seconds 12. The distance between the sacral promontory and the posterior superior border of the

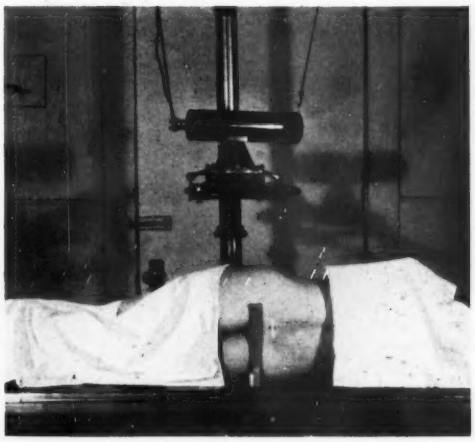

Fig. 8. Patient in position for lateral pelvimetry. The distance from the spinous process of the fourth lumbar and the symphysis pubis to the film must be equal to insure parallelism of the conjugata vera and film.

symphysis pubis is measured and corrected on Walton's chart.

COMPARATIVE DATA

One hundred women were examined roentgenologically in the X-ray Department of

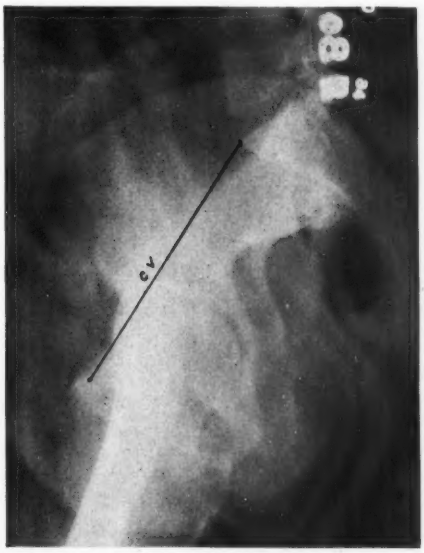

Fig. 9. Film obtained with patient in position shown in Figure 8. The magnified conjugata vera is shown; this is corrected as explained in Figure 7.

TABLE I.—COMPARATIVE MEASUREMENTS OF EACH CASE

Case	Anteroposterior diameter—conjugata vera			Transverse diameter	
	Walton	Moloy	Lateral view	Walton	Moloy
1	10.7	10.5	..	11.6	11.2
2	7.5	7.0	..	12.3	11.5
3	11.6	11.6	..	12.0	12.2
4	11.2	11.0	..	12.1	12.3
5	10.7	9.1	..	13.2	11.8
6	9.7	9.8	..	11.5	11.6
7	11.0	11.1	..	13.6	13.6
8	11.1	11.1	..	10.9	11.5
9	10.7	10.2	..	13.0	12.1
10	11.3	12.2	..	11.8	11.3
11	10.9	9.5	..	12.3	11.1
12	11.6	11.4	..	12.4	12.5
13	11.3	11.4	..	12.5	12.5
14	11.9	12.2	..	12.1	12.5
15	12.5	12.0	..	14.1	13.5
16	10.3	10.8	..	12.3	12.3
17	11.3	12.2	..	13.6	14.4
18	12.0	10.9	..	13.4	12.5
19	10.9	9.6	..	12.2	11.7
20	9.5	9.8	..	12.0	12.2
21	10.2	10.1	..	11.2	11.8
22	11.8	11.5	..	12.2	11.7
23	11.3	11.0	..	11.5	11.5
24	11.0	11.2	..	12.5	12.6
25	10.8	11.1	..	11.8	12.2
26	10.1	8.5	..	11.8	11.4
27	12.0	11.5	..	12.3	11.7
28	9.7	10.5	..	10.3	11.2
29	9.2	9.1	..	12.5	12.2
30	6.7	6.7	..	11.4	11.5
31	11.1	10.8	..	11.8	11.5
32	10.3	10.2	..	12.2	12.3
33	10.2	9.8	..	10.6	10.1
34	11.6	11.7	..	12.4	12.0
35	10.9	9.0	..	11.8	10.5
36	9.6	9.5	..	12.7	12.7
37	11.0	11.4	..	12.0	12.0
38	9.6	9.2	..	12.3	12.2
39	9.6	9.0	..	11.9	11.5
40	12.5	12.4	..	13.5	14.0
41	9.7	9.1	..	12.4	12.2
42	9.6	10.2	..	12.1	13.0
43	13.3	13.4	..	13.3	14.0
44	12.0	12.1	12.4	11.1	11.1
45	10.6	12.0	11.0	12.4	13.4
46	9.9	9.9	9.8	10.2	10.2
47	10.8	10.8	10.8	12.6	12.4
48	12.6	12.8	13.0	12.0	12.2
49	10.4	9.9	10.3	11.8	12.5
50	11.9	11.6	11.9	12.8	13.0
51	10.7	10.5	10.0	13.7	14.0
52	10.5	9.9	10.5	13.3	12.9
53	10.1	9.7	10.1	11.7	11.4
54	11.6	11.0	12.0	13.9	13.7
55	10.5	8.2	10.4	11.7	11.0
56	10.2	10.2	10.2	12.7	13.2
57	11.8	12.2	12.3	12.0	11.9
58	11.6	11.2	12.2	12.6	12.6
59	11.7	11.5	11.7	13.2	13.8
60	10.3	9.5	10.3	11.3	12.8
61	10.8	9.9	10.9	11.9	11.2
62	11.0	10.3	11.1	12.4	12.2
63	11.6	11.2	11.4	12.7	12.1
64	11.9	11.9	12.0	11.7	11.2
65	9.3	9.0	9.4	11.5	11.9

TABLE I.—COMPARATIVE MEASUREMENTS OF EACH CASE—Continued

Case	Anteroposterior diameter—conjugata vera			Transverse diameter	
	Walton	Moloy	Lateral view	Walton	Moloy
66	10.9	11.8	10.9	11.4	11.9
67	11.9	11.9	11.9	12.6	12.6
68	9.9	9.1	10.0	13.7	13.1
69	8.8	8.5	10.2	12.2	12.2
70	11.4	11.9	11.7	13.5	14.1
71	11.8	12.0	12.0	12.3	12.2
72	10.8	11.1	11.0	11.9	12.1
73	12.5	12.0	12.6	12.8	12.0
74	12.7	12.5	13.0	12.3	13.4
75	10.8	10.6	10.8	11.0	10.8
76	11.6	12.0	11.2	11.3	11.2
77	11.3	12.1	11.6	11.7	12.8
78	12.2	12.2	12.1	12.5	12.7
79	11.8	12.2	11.7	12.9	13.0
80	9.8	9.8	9.6	10.7	11.2
81	9.0	9.1	9.0	13.2	13.2
82	10.0	9.2	9.9	12.4	12.6
83	10.3	10.1	10.6	12.5	12.5
84	12.3	12.2	12.6	13.2	13.2
85	12.5	12.6	12.8	13.3	13.0
86	9.2	10.2	9.1	13.0	12.8
87	10.6	10.2	10.5	14.0	14.1
88	11.9	12.1	11.8	13.4	13.6
89	10.2	9.8	10.3	12.7	12.1
90	10.5	10.1	9.9	12.3	12.0
91	12.0	11.7	12.0	12.9	12.8
92	10.4	10.6	10.6	13.8	14.4
93	10.4	11.2	10.3	12.7	13.0
94	11.6	11.9	12.0	12.8	12.5
95	10.3	10.3	10.4	12.8	12.7
96	10.9	11.0	10.7	11.7	11.7
97	9.9	9.7	9.7	12.2	12.2
98	11.3	11.3	11.1	11.6	12.0
99	9.3	9.1	9.3	12.8	13.0
100	9.3	9.2	9.2	12.9	13.2

Bellevue Hospital between August, 1934, and April, 1935. The first 43 cases were examined by the Moloy stereoscopic and Walton anteroposterior methods. The last 57 cases were examined also in the lateral position, with the Walton chart for measurements. In Table I the measurements obtained in each case for the conjugata vera and transverse diameter of the superior strait are tabulated. Table II summarizes the discrepancies in measurements between the methods. It is seen that 73 per cent of the cases show no more than 0.5 centimeter difference in the transverse diameter measurement. The differences cited in the tables may be due to variation either way—the Moloy method occasionally reading more and occasionally less than the Walton method. Twenty per cent of cases showed a difference in reading of 0.5 centimeter in the transverse diameter. In the measurement of the conjugata vera, the Walton antero-

TABLE II.—SUMMARY OF AMOUNT OF VARIATION IN READINGS OF ANTEROPOSTERIOR AND TRANSVERSE DIAMETERS FOR EACH OF THE METHODS USED

Diameter being measured	Conjugata Vera									Transverse diameter		
Methods being compared	Anteroposterior (Walton) Moloy stereoscopic			Anteroposterior (Walton) Lateral (Walton)			Moloy stereoscopic Lateral (Walton)			Moloy stereoscopic anteroposterior (Walton)		
Differences in centimeters	No. of cases	Total cases	Per cent	No. of cases	Total cases	Per cent	No. of cases	Total cases	Per cent	No. of cases	Total cases	Per cent
.0	12	75	75	13	53	93	7	40	70	16	73	73
.1	17			19			7			13		
.2	15			8			10			16		
.3	12			6			6			9		
.4	11			6			4			10		
.5	8			1			6			9		
.6	6	18	18	2	3	5	2	15	26	8	20	20
.7	1			1			1			4		
.8	6			0			3			3		
.9	4			0			5			4		
1.0	1			0			4			1		
Over 1 cm.	7		7	1		2	2		4	7		7

posterior and Moloy stereoscopic methods checked within 0.5 centimeter in 75 per cent of the cases and between 0.6 and 1 centimeter in 18 per cent of the cases. The lateral pelvic mensuration checked with the Walton anteroposterior method for a difference of 0.5 centimeter or less in 93 per cent of the cases. In only 1 case did these two methods show a variation greater than 0.7 centimeter. The lateral plate measurements compared to the Moloy measurements about as the anteroposterior (Walton) plates did (see Table II). In 7 cases discrepancies were shown of more than 1 centimeter but 5 of these 7 were among the first in which measurements were made. As our technique improved the figures obtained by all methods agreed more closely. The fact that the lateral pelvic measurements of the conjugata vera checked so closely with the anteroposterior film measurements in so many cases indicates only that our technique in carrying out these two methods of geometrical measurement is fairly accurate. However, when these two figures do compare closely and yet are at a considerable variance with the stereoscopic measurement, we are inclined to favor the former as being somewhat more accurate.

CONCLUSIONS

In this communication, we have attempted to set forth the principle and technique of the methods we selected for a study of pelvimetry. We may conclude that the methods of anteroposterior pelvimetry and lateral pelvimetry described are simple procedures and give results which agree closely with each other and with those of the stereoscopic method. The reading of the stereoscopic plates according to the method described requires some experience and, of course, the prism stereoscope. There is no reason to believe that the measurements obtained with the stereoscope are more accurate than those obtained by the other methods described. However, this publication deals only with pelvimetry and if the fetal head-bony canal relationship is being investigated the stereoscope is very valuable.

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THE ESTROGENIC PRINCIPLE, THE COMMON ETIOLOGICAL FACTOR OF ENDOMETRIAL HYPERPLASIA, UTERINE FIBROIDS AND ENDOMETRIOMAS

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ENDOMETRIAL hyperplasia, uterine fibroids, and endometriomas are three of the very common conditions encountered in the practice of gynecology. All three have been dealt with rather extensively in medical literature, yet only has the etiology of endometrial hyperplasia been generally accepted. The cause of the two tumors is still vague and only general explanations are offered as to their etiology. That these three conditions could possibly have a common etiological background has only recently been entering into medical literature; however, it is the hypothesis of this paper that such is the case, and that the fundamental cause has its origin in excessive stimulation by the ovarian follicular hormone on the endometrium, myometrium and the ectopic locations from which the endometriomas arise. That the action of this hormone is not specific to the uterine endometrium alone, but acts upon the genital tract as a whole, is easily demonstrated (38). When this action on the endometrium is abnormal, causing endometrial hyperplasia, it is equally abnormal in its action upon the myometrium, causing by cellular metaplasia of the uterine muscle cell or cells, the subsequent development of uterine fibroids. Likewise irregularity of action of this hormone on potential serosal cells, by cellular metaplasia of these cells, or by tumor proliferation of aberrant endometrial implants, causes the formation of endometriomas.

ENDOMETRIAL HYPERPLASIA

The subject of this paper naturally divides itself into 3 parts, and each will be discussed separately. Endometrial hyperplasia is a very common and important gynecological condition. It is characterized by profuse and irregular bleeding from the uterus, hyperplasia of the endometrium of the "Swiss cheese" pattern, excess ovarian follicular hormone in the blood and urine, multiple follicle cysts of the ovary and absence of corpora lutea. From the work of Schroeder and Meyer in Germany; Shaw in England; Graves (14), Fluhman (12), Novak and Martzloff, and Burch in this country, the cause of endometrial hyperplasia has been thoroughly investigated. That the persistent and excessive ovarian follicular hormone stimulation, in the absence of any corpora lutea and with a possible anterior hypophyseal action in the background, is the cause of endometrial hyperplasia has well been established by these observers. In fact, Burch (5) and his co-workers have produced endometrial hyperplasia in spayed animals by the injections of the estrogenic principle. The general acceptance is, at present, that endometrial hyperplasia is the result of the unopposed and continued action of an excess amount of the estrogenic principle derived from the multiple follicle cysts of the ovary.

UTERINE FIBROIDS

Histogenesis. What is the cause of uterine fibroids is a question that is asked daily of the gynecologist and one which he can at present answer only in vague generalities. The histogenetic study of fibroids does not reveal in the uterine muscle any certain distinctive myoma mother cells from which might be traced the stages that lead to a proliferating myoma. Robert Meyer (23), Becher, and Heimann have practically settled the question of the histogenesis of uterine fibroids. They have studied in serial sections the smallest myomatous tumors and have found that the earliest appearance of a myoma represents a thickening of the uterine muscle bundles which are directly connected with the normal musculature of the uterus. These tumors are generally devoid of blood vessels and therefore their origin cannot be ascribed to this source, but from the normal uterine muscle itself (15).

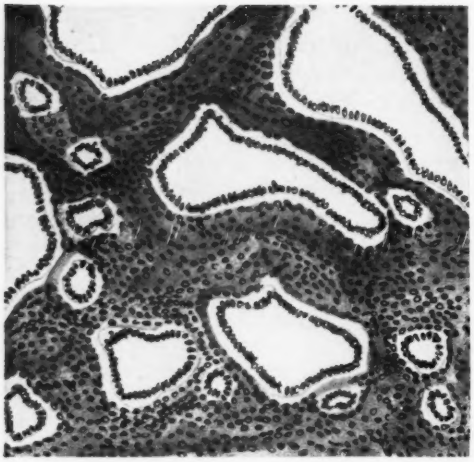

Fig. 1. Hyperplasia of the endometrium of the uterine mucosa.

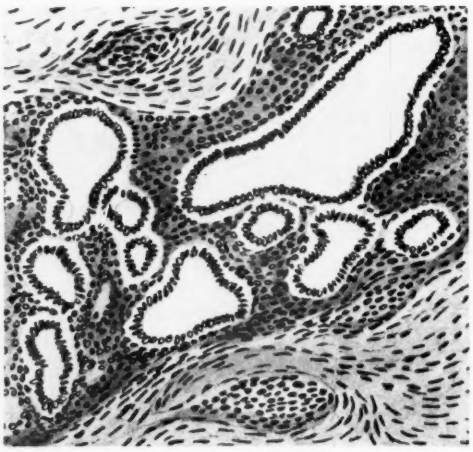

Fig. 2. Endometrial hyperplasia of an ovarian endometrioma. Both slides were taken from the same patient.

On the other hand DeSnoo suggests that the uterus is a very special primitive organ, an organ having a great many undifferentiated cells, genitoblasts, which are of very great physiological importance. While these cells in the embryonic life form the uterus proper, later on they serve to provide material for the growth of the uterus during pregnancy and for the regeneration of the endometrium during the puerperium. Under pathological conditions these genitoblasts can give rise to the formation of uterine fibroids, adenomyomas, and endometriomas. The remarkable thing about these genitoblasts is that, both under physiological and pathological conditions, their activity and proliferation depend entirely upon the functioning of living ovarian tissue.

It must be admitted, however, that the ultimate causation of uterine fibroids is as far from being explained as that of other new-growths, but the hypothesis here offered is that excessive ovarian follicular hormone stimulation is the igniting factor that causes cellular metaplasia of a certain uterine muscle cell or cells which, if the hormonal stimulation continues, will develop into uterine fibroids.

The exact nature of the action of the estrogenic principle upon uterine tissue is still only partially understood. If hormones are metabolic products of cells, secreted into the blood stream, it would seem logical to look for action directly upon the cells involved. Opposed to this view is the hormonal stimulation of the vascular control mechanism. There can be little question that this mechanism is involved in some way, due to the hyperemic conditions of the organs stimulated (1).

ETIOLOGY

Graves (15) has aptly drawn attention that uterine fibroids possess certain attributes that seem to place them in a borderland between physiological hypertrophy and true neoplastic growths. "Their great frequency (20 per cent of all women over 35), their apparent coincidence with the sexual era, their regression after ablation of the ovarian function, their histological similarity to diffuse hypertrophy and gestation hyperplasia, their structural identity with the normal musculature, are all familiar facts that lend to the tumors a physiological glamour which they probably do not deserve."

In determining the ultimate cause of fibroids, it must be considered first whether or not uterine muscle cells possess an inherent growth capacity different from that of smooth muscle in other parts of the body. That such a fact is true is well demonstrated by clinical and histological observations of diffuse uterine

hypertrophy in conditions of "fibrosis uteri" and in gestational hyperplasia. Second, it must be determined whether the uterine muscle is subjected to a specific external stimulus that is able to call forth this growth response. That such a specific stimulus is present is readily shown by the growth action of the estrogenic principle on the myometrium. Clauberg has published roentgenograms demonstrating by means of lipiodol injections the greatly increased size of the cavity of the uterus in a castrated woman after large injections of the follicular hormone. An unlimited number of observers have noted the same effect on animal uteri after the administration of the same hormone. The uterus becomes enormously enlarged because of its rapid growth and increased vascularity—an effect due solely to the hormone elaborated by the graafian follicle (Van Herweden and Corner).

The popular theories of etiology of uterine fibroids are: (1) irritation theory—the irritating stimulus of infection stimulates abnormal growth. (2) Circulatory theory—the hyperemic condition of the uterus during pregnancy and the puerperium, when hyperemia is at its peak, rules out this theory. (3) Growth-energy theory—this theory represents an unsatisfied tendency of the uterine muscle to hyperplasia, which finds its natural expression in the enormous growth of pregnancy. When pregnancy does not occur, the uterus manifests an abnormal proliferation in the nature of fibromyomatous growths. (4) Sterility—patients with fibroids exhibit sterility 3 to 4 times as frequently as normal women. Whether sterility is the cause of fibroids or fibroids the cause of sterility is the essential problem. (5) Heredity—occasionally sisters of one family, or mother and daughter will present fibroids, although only 21 women in Lynch's 683 cases gave an indicative family history. (6) Race—fibroids are exhibited about 9 times as frequently in the colored woman as in the white. Such an observation is of interest because of the rarity of this condition in the primitive African savages. The increasing civilization of the Negro woman has inflicted this acquired condition upon her (36).

Fibroids are comparatively unknown in the lower animals. Sampson suggests such findings are due to lack of true menstruation in these animals, while DeSnoo goes back one step further and ascribes their prevalence in woman to be due to the stronger ovarian activity in humans.

The present tendency is to explain the cause of uterine fibroids on some irregularity of function of the hormones that regulate the growth and physiology of the pelvic organs. However, it must be remembered at the onset that this dysfunction, which is mainly hypophyseal or ovarian, acts as a sexual growth stimulant on the entire genital tract. Not only is endometrial hyperplasia induced by such dysfunction, but the tubal muscularis and mucosa, the myometrium, the vagina, vulva, and breasts are all stimulated to increased mitotic cellular division and hypertrophy. In fact, this estrogenic principle is a growth stimulant to the entire mullerian tract, and the development of abnormal conditions, such as endometrial hyperplasia due to its over action is a manifestation of its dysfunctional action in only one locality. The myometrium, the tubes, aberrant endometrium, the breasts or any part of the genital tract can likewise express abnormal lesions of growth resulting from its dysfunctional stimulation.

In three former communications (34, 35, 36) the rôle of multiple follicular cysts of the ovary, resulting in hyperestrinism, which stimulates the uterine endometrium to an abnormal hyperplastic condition, was discussed. It was hypothesized that this abnormal estrogenic stimulus affected not only the endometrium, but the myometrium as well. Since fibroids tend to grow slowly, this abnormal estrogenic stimulus would produce immediate hyperplasia of the endometrium and gradually developing fibroids, provided the stimulus was prolonged sufficiently. Such an explanation can easily account for the great prevalence of fibroids in the negro race. The enormous incidence of pelvic inflammatory disease in the negro produces pathological changes in and around the ovaries, resulting in permanent ovarian dysfunction, which will express itself immediately as endometrial hyperplasia, and more latently as fibromyomatous changes in the myometrium.

This hypothesis has been recently supported by King, who states: "Another fact of interest bearing on the clinical side of endometrial hyperplasia is its frequent association with fibroid tumors. Its frequency would surely exclude its being a chance relationship. It is more commonly found in connection with the smaller tumors, as the large ones, by stretching and pressure, are more likely to produce endometrial atrophy. In 114 cases of fibroids, 71 per cent of the cases exhibited hyperplasia of the endometrium. A further suggestive point is found in the frequent combination of endometrial and myometrial hyperplasia."

Allen (2) also lends support to this theory of origin of uterine fibroids. The high incidence of fibromyomas associated with endometrial hyperplasia suggests to Allen the inclusion of these masses of functionless tissue as an end-result of hormonal stimulation, another type of cellular metaplasia caused by underlying glandular dysfunction. Such a postulate, this author observes, has many interesting and new angles. If accepted as a starting point, it will lead into the field of the cause of all benign tumors, while further metaplasia or dedifferentiation carries on directly into the realm of malignancy.

Lewis and Geschickter have shown the presence of the gonadotropic and estrogenic principles in a myoma of the uterus, while Zondek and Fluhman (11) have demonstrated a measurable amount of prolan A in the urine of about one third of their cases of fibroids (Allen, 2).

CLINICAL DATA

The present paper is 2 years' additional evidence in support of a cause and effect relationship between multiple follicle cysts of the ovary, with excess estrogenic hormone secretion, immediate production of endometrial hyperplasia, and more latent development of uterine fibroids, if the stimulation is sufficiently prolonged.

Forty-four cases of endometrial hyperplasia were studied. On each patient a curettage was performed and the curettements diagnosed microscopically as hyperplasia of the endometrium. In no case, either by bimanual examination or with the curette, was a uterine fibroid noted. In addition, 20 of the cases (45 per cent) had a laparotomy performed and in no instance was a fibroid found, but in every case multiple follicle cysts of the ovaries were observed. After varying intervals, the average being 4 years and 9 months, all 44 patients returned for a second operation because of uterine fibroids, and the findings of the endometrium, myometrium, and ovaries at the time of the second operation, are here offered as evidence of an interrelationship between multiple follicle cysts of the ovary, endometrial hyperplasia and uterine fibroids.

ANALYSIS OF TABLE I

The age limits were well within woman's functional years, 30 to 39 years accounting for 77 per cent of all patients, the average being 36 plus years. The symptoms between the first and second operations offer an interesting comparison, the outstanding feature of which was an increase of complaints. Radium caused a temporary amenorrhea in 2 cases, while in 6 cases no benefit was derived from its application. Pregnancy occurred in 2 women after the initial curettage. The average time between the two operations was 4 years and 9 months. The abdomen was opened in 20 patients in addition to the 44 curettages, and in 16 instances of the 20 cases the ovaries were diagnosed, either microscopically or operatively, as containing multiple follicle cysts. The amount of the curettements was described, but since different doctors performed the curettages, the description naturally varies with the operator.

At the second operation the uterus was removed in every instance because of fibroids. Both ovaries were excised in 18 cases, while all the remaining ovarian tissue was removed in 10, a total of 64 per cent. Unless both ovaries or the total ovarian tissue are studied in their entirety, it is unwise to draw conclusions concerning the presence or absence of the corpus luteum, since its absence is so importantly related to hyperplasia of the endometrium. In 4 cases developing corpora lutea were found with associated premenstrual endometrium, while in 35 cases no ovarian yellow body was observed. Multiple follicle cysts of the ovaries were present in all 44

cases, including those in which the corpora lutea were found. Any explanation for the coexistence of follicle cysts of the ovaries and corpora lutea must be in the nature of an hypothesis. That endometrial hyperplasia is not always a permanent condition is frequently seen just after the puberty age when menstruation, disturbed at first, settles down to its normal rhythm. During the transition period between abnormal functional bleeding to normal menstruation, the existence of multiple follicle cysts and corpora lutea might be observed.

The myometrium was hyperplastic in 24 cases, 55 per cent, and was adenomyomatous in 8. Salpingitis and adhesions were noted 38 times, 86 per cent, while ovarian endometrial transplants were present in 22 cases, or 50 per cent. Combining this latter figure with the adenomyomatous findings, endometriomas were found to be present in 28 patients, 64 per cent. Two patients exhibited combined myometrial and ovarian endometriomas. This observation will be discussed subsequently in more detail.

DEDUCTIONS

These 44 cases of endometrial hyperplasia, on which a second operation was performed on an average of 4 years and 9 months later for fibromyomatous growths of the uterus, are convincing evidence of a cause and effect relationship of the prolonged and unopposed action of the ovarian follicular hormone on the myometrium and the subsequent development of uterine fibroids. Evidence has been sufficiently advanced for the general acceptance of hyperestrin stimulation of the ovary from multiple follicle cysts as the cause of endometrial hyperplasia. The excess of action of this hormone or its dysfunction is not limited solely to the endometrium; all of the genital tract is stimulated to hypertrophy and hyperplasia by its action. The contention of this paper is that when this hormonal stimulation is of sufficient strength or when its action is unopposed over a lengthy period, it is the igniting factor which causes cellular metaplasia of one or of several of the uterine muscle cells, with the subsequent development of uterine fibroids.

TABLE I

	Cases
Type of first operation	
Dilatation and curettage	44
Application of radium	8†
Suspension of uterus	10*
Unilateral salpingo-oophorectomy	10*
*Abdomen opened in 20 cases, 45 per cent of total. †Number of hours undetermined.	
Condition of ovaries at first operation	
Diagnosed microscopically as cystic	10
Diagnosed operatively as cystic	6
Not mentioned at operation	4
Curettements	
Abundant	16
Polypoid	8
Moderate	8
Small	5
Not mentioned	7
Time interval between operations	
Longest	13 years, 2 months
Average	4 years, 9 months
Shortest	1 year, 2 months
Type of second operation	
Hysterectomy	44
Bilateral salpingo-oophorectomy	18*
Removal of all remaining ovarian tissue	10*
Unilateral salpingo-oophorectomy	16
*All ovarian tissue removed in 28 cases, 64 per cent of total.	
	Cases
Condition of myometrium (microscopic)	
Fibromyomatous	44
Hyperplastic	24
Fibrotic	6
Adenomyomatous	8
Normal	14
Malignant	1
Condition of endometrium (microscopic)	
Hyperplastic	40
Premenstrual	4
Condition of ovaries at second operation (microscopic)	
Multiple follicle cysts	44
Corpus Luteum	
Mature	4
Degenerating	5
Absent	35
Miscellaneous	
Salpingitis	18*
Adhesions	20*
Ovarian endometrial transplants	22†
*38 cases, 86 per cent of total. †50 per cent of total.	

ENDOMETRIOMAS

Endometriosis is a term introduced by Sampson to include a variety of adenomatous lesions of the female pelvis, the histological and functional characteristics of which are identical with those of the endometrium. The

etiology of endometriomas is still a matter of debate. Up to the present we must offer thanks (1) to Cullen for the mucosal or diverticular origin of the diffuse uterine growths; (2) to Ivanoff for his serosal theory, which draws attention to the proliferative potentialities of the peritoneal mesothelium; (3) to Sampson (26) for the hypothesis of tubal regurgitation or retrograde menstruation theory with direct implantation of living endometrial grafts; and (4) to Robert Meyer (24), championed in this country by Novak, for his invaluable demonstrations of epithelial heterotopy and metaplasia which helps to render more intelligible many extrapelvic growths of like character. Suffice it to say that at present two views are generally held as the cause of these tumors, either the cellular spill hypothesis of Sampson or the theory of serosal cell heteroplasia advocated as early as 1898 by Ivanoff. That neither one of these theories can explain the occurrence of endometriomas at all sites is generally accepted. The heteroplasia view of the serosal cell cannot explain the perineal endometriomas, while inguinal canal endometriosis is difficult to ascribe to the implantation theory of fragments of the uterine lining.

It is not the intention of this paper to enter this field of discussion, and the following remarks and observations will hold good irrespective of the primary source of the endometrial elements. If the endometrioma is explained by the implantation view, there must be some stimulant or environment which decides whether the endometrioma will grow or perish. Sampson (27) took cognizance of this fact and stated that "the escaping cells need suitable soil." On the other hand, if the origin of the endometrioma is from heteroplasia of serosal or germinal cells, some factor must be present which first instigates the cellular change and which subsequently stimulates the endometrial growth. The actual origin of the endometrioma is unimportant, whether from implantation of a living graft or from cellular metaplasia; the all important question is the determination of the cause of the igniting factor which controls this cellular change or which stimulates to proliferation the endometrial implants.

The morphological and functional characteristics of endometriomas are similar to uterine endometrium; its integrity and function are dependent upon the presence of active ovarian tissue, since castration causes regression of the tumor; it presents decidual reaction during pregnancy; it undergoes the phases of the menstrual cycle, changes dependent on the ovarian hormones; and Gleave has proved that in rabbits the presence of the estrogenic principle is essential for the maintenance of the lesion.

Since the ovarian follicular hormone is the cause of endometrial hyperplasia and the histological structure of endometriomas and uterine endometrium are similar, it is logical to deduce that the igniting factor of endometriomas, which brings about the cellular metaplasia or the implant proliferation, is the estrogenic principle. That such is the case is all the more established by the fact that many endometriomas present histologically endometrial hyperplasia, and also by the high incidence of association of endometriomas with uterine endometrial hyperplasia. The frequent finding of all the features of endometrial hyperplasia in the endometriomas, accompanied by similar changes in the uterine mucosa, can only be caused by the factor which determines the latter, the ovarian follicular hormone.

In a former contribution (35) while studying the relationship between endometrial hyperplasia and uterine fibroids, the high incidence, 30 per cent, of ovarian endometrial transplants was noted. At that time the suggestion was made that these three conditions might possibly have a common etiological background. These figures have been rechecked and, together with the findings in the present paper, ovarian and uterine endometriomas were associated with endometrial hyperplasia and uterine fibroids in 64 per cent of the cases, a figure far too high to make such a finding mere co-incidence.

Other authors have likewise called attention to the high incidence of association of these three conditions. In Jeffcoate's series of 113 cases of endometriomas, 79 women, 71 per cent, exhibited endometrial hyperplasia, while 31, or 28 per cent, presented

fibroids in the uterus. In discussing endometriosis, Allen (2) was impressed with the high incidence of association of this condition with menstrual irregularities due to endometrial hyperplasia, 70 per cent, uterine fibroids, 41 per cent, and the prevalence of relative sterility, 60 per cent. The marked irregularities of the menstrual habit immediately suggested a glandular imbalance. He considered that such a hypothesis more accurately explained those instances of irregular bleeding occurring in patients in whom the endometriomas were so located that they could not possibly have caused the hemorrhage. Smith, in 159 cases of endometriomas, noted endometrial hyperplasia associated in 42 per cent and uterine fibroids in 52 per cent of the women. Other observers, Taylor, Shaw, Beckman, Novak and Martzloff, and Tietze have reported the occurrence of these stigmas of glandular activity in patients afflicted with endometriosis.

Therefore, it seems logical to deduce that the multiple follicle cysts of the ovaries, in the absence of corpora lutea, which cause, through the action of the estrogenic principle, endometrial hyperplasia, likewise cause the ectopic endometriomas. Figures 1 and 2 are presented to show the similarity of the hyperplasia of the endometriums of the uterus and ovarian endometrioma. Both slides were taken from the same patient.

There are two clinical features common to these three conditions, uterine hemorrhage and absolute or relative sterility. Functional uterine bleeding has long been observed as the clinical manifestation of endometrial hyperplasia. Graves (14) and King have called attention to this same clinical feature in women with uterine fibroids which were so located that the origin of the bleeding could not possibly have come from the fibroids. Their explanations are that the bleeding results from the associated endometrial hyperplasia. Likewise Allen (2) offers endometrial hyperplasia as the source of the uterine hemorrhages in women with endometriomas so situated that they could not have been the cause of the bleeding.

Sterility absolute or relative, is another clinical feature characteristic of these three conditions. In endometrial hyperplasia, when the patient is bleeding, absolute sterility is assured. In women with fibroids, sterility runs as high as 35 to 40 per cent of the cases, while in cases of endometriomas Herd and Allen (2) place the figure around 60 per cent. The explanation for the sterility in these three conditions is undoubtedly lack of ovulation, so well demonstrated in the ovaries by the presence of multiple follicle cysts and the absence of corpora lutea.

CONCLUSION

The hypothesis that all forms of overgrowth of the uterine endometrium or musculature are due to the same factor, the estrogenic principle, is not only supported by clinical and pathological data, but also explains satisfactorily the simultaneous development of endometrial hyperplasia, endometriomas and uterine fibroids, with their associated clinical features, hemorrhage and sterility.

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HISTOLOGICAL STUDIES OF ENDOMETRIUM DURING VARIOUS PHASES OF MENSTRUAL CYCLE[1]

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WHILE the essential features of the menstrual cycle have been recorded by many observers, there is a distinct lack of correlation between the observed state of the endometrium and the clinical picture. Further, no unity exists in the present system of reporting the several phases of the cycle. The object of this study was to attempt to clarify this seemingly chaotic state and to present some of the newer conceptions regarding the regenerative phenomena that occur in the menstrual cycle.

Present knowledge concerning the menstrual cycle may be stated briefly as follows: Westphalen, in 1896, was probably the first to study the endometrium in relation to the menstrual cycle. Although his work was done with material obtained at necropsy and under some technical handicaps, it nevertheless was of some consequence. Westphalen believed that regeneration of tissue was complete by the fourteenth to fifteenth day; he did not, however, give any results to support this belief.

The next notable contribution was made in 1908 by Hitschmann and Adler who studied uteri removed during the menstrual cycle. Their work is accepted today, with some modifications; their records, however, were incomplete, especially regarding the menstruating or "tissue loss" phase, and this information was supplied by the excellent observations of Schroeder in 1915. Novak and TeLinde (22), in 1924, reported a study corroborating the results of Schroeder. The material for this study was collected in such a way as to eliminate possible errors of technique which had been responsible for misinterpretation in many previous studies. The outcome of these observations was the classification of the cyclic changes in the endometrium as follows: (1) postmenstrual phase, (2) interval phase (called by some, resting endometrium), (3) pregravid or premenstrual phase, and (4) menstruating endometrium. This classification is accepted and used by most workers in this country at the present time. Novak (23) subscribed to this classification and recently has described each phase.

A few significant facts concerning the nature of the process under study also deserve mention. Zeleny, in 1916, in a study of regenerative phenomena under normal conditions, observed certain phases: (1) a phase of regulation, which was characterized by reorganization and migration of cells in the area of "tissue loss," (2) a phase of cellular proliferation, which was characterized by mitosis and a rapid increase in the size of the regenerating area, and (3) a phase of cell and tissue differentiation, which was accompanied by decline in cellular proliferation and by an increase in the size of the regenerating area. The regulative phase lasts for approximately 48 hours; the proliferative phase then follows and reaches a maximum in 7 to 10 days; this is then followed by the differentiative phase, which reaches a maximum in about 18 days, when the increase in size of the regenerating area has about ceased. Differentiation is characterized by the change into the definitive or functioning tissue. This work was checked by Speidel, in 1929, and he observed the same phases. In 1933, Herrell observed similar results in the regeneration of tissue as a result of pituitary stimulus, and while these studies were not of human tissues, it seemed to us that the same basic principles might well hold for regenerating tissues of any type. With this in mind, we at once sought a human tissue suitable for such studies. Interestingly enough, the endometrium is unique in that it, in a cyclic fashion, undergoes "tissue loss" which is followed by the phenomenon of complete regeneration.

[1] Abridgment of thesis submitted by Dr. Wallace E. Herrell to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery.

With these facts in mind, the present study was undertaken to apply these principles of regeneration, if possible, and to correlate the observed states of the endometrium with the basic physiological principle at work in the process.

MATERIAL AND METHOD

Material for this study was obtained from the operating rooms of the Colonial Hospital and it was examined in the laboratory of surgical pathology under the supervision of Dr. Broders. Portions of more than 241 specimens of endometrium were prepared by the fresh frozen section method of Wilson, as modified by Broders, and such specimens were stained with Terry's neutral polychrome methylene blue. Portions of 160 of these specimens of endometrium were prepared for study by the fixed frozen section technique, and these were stained with hematoxylin and eosin. Data were obtained in each case from the clinical record and from the individual patient. By this means, specimens were observed during every phase of the menstrual cycle, and they were obtained from whole uteri or from scrapings secured in the course of dilatation and curettage. Fifty-two specimens were obtained by the use of the instrument recently described by Randall.

EXPERIMENTS AND OBSERVATIONS: PHASES OF THE CYCLE

Menstruating phase. Five specimens were obtained during the phase of "tissue loss," and according to the observations made there can be no doubt that loss of tissue is complete in the first 24 hours of menstruation and that the remainder of the menstrual period is one of hemorrhage and secretion. This observation is in accord with observations made by Novak and others. Some observers, however, do not concur in this view. Figure 1 illustrates this "tissue loss." The specimen was obtained 24 hours after the onset of the menstrual period of a patient whose cycle may be considered normal. The loss of tissue corresponds approximately to three-fourths of the endometrium; there remains only the basal layer of endometrium, which measures approximately 0.5 millimeters. This picture might well be confused with that of a freshly curetted uterus. The glands in the basal layer remain essentially the same throughout the cycle; hence, one can learn very little concerning the phase from such specimens when obtained either by dilatation and curettage or from the whole uterus. The surface and outer layer of endometrium, however, afford means of determining the phase of the menstrual cycle accurately, although this is as easy to determine from specimens removed at dilatation and curettage as it is from a block from the whole uterus, assuming the sections are prepared properly. The small glands, which will be seen below the basal layer of endometrium, should be noted (Fig. 1). Of particular significance also is the height of the epithelium and the general configuration of these glands. Figure 2 illustrates the difference between the histological structure of the basal epithelium and the epithelium of these glands. The latter epithelium remains essentially the same throughout the cycle. The term "subbasal" has been proposed for these glands, to which reference will be made later in this paper.

Early reparative phase of migration and rearrangement of cells. The next 24 hours of the menstrual cycle are taken up with reorganization, rearrangement, and migration of cells. From microscopic study, one is able to learn that the cells lining the remaining glands will take part in resurfacing the endometrium. Mitosis is not active; in fact, this phase is similar to the early phases observed before by Zeleny, Speidel, and Herrell, in the study of regenerating tissues. No evidence has been collected in this study in support of the theory of metaplasia of the stroma in resurfacing endometrium. In fact, glandular tissue seems the most likely source of the resurfacing epithelium.

Early proliferative phase (first to seventh day). This phase corresponds roughly to that previously and, we believe, incorrectly, called the postmenstrual phase. It is characterized by active division of cells, resurfacing of the endometrium, and formation of new, straight tubular glands from the surface epithelium (Fig. 3). Mitosis is active, and cellular proliferation occurs also in the loose embryonal

Fig. 1. Menstruating phase ("tissue loss"). Specimen obtained 24 hours after onset of menses in a normal cycle. Note subbasal glands and loss of outer two layers of endometrium. Stained with hematoxylin and eosin (×35).

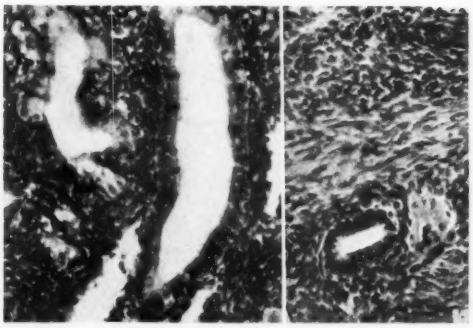

Fig. 2. Left, a photomicrograph of the epithelium of the basal gland at a high magnification to be compared with figure at right. This illustrates typical late differentiative phase in contrast to the inactive epithelium of the subbasal gland. Stained with hematoxylin and eosin (×140). Right, Subbasal gland. Photomicrograph at same magnification as figure at left to illustrate difference in height of epithelium.

type of stroma. By the end of the first week in this phase, the endometrium presents a fairly typical picture. The average number of glands per low power field is three to four, and these glands are nearly straight tubules, the epithelium of which is of moderately low columnar type, with nuclei situated near the center of the cell. The endometrium grossly is from 1 to 1+ millimeter in thickness. One may therefore at once state the time of the last period, and hence the phase of the new cycle. One should always study these glands, if possible, in the longitudinal view; if they are seen on cross section, however, the phase is discernible by the characteristics just outlined. The tubular glands will appear in cross section merely as small circles, with other features of the early proliferative phase (Fig. 4).

Late proliferative phase (eighth to fourteenth day). This phase corresponds roughly to what has previously been called interval or resting endometrium, and it is with this phase that we especially wish to deal. The regenerative phase here is *not* a resting endometrium, nor is there such a phase. The very nature of the

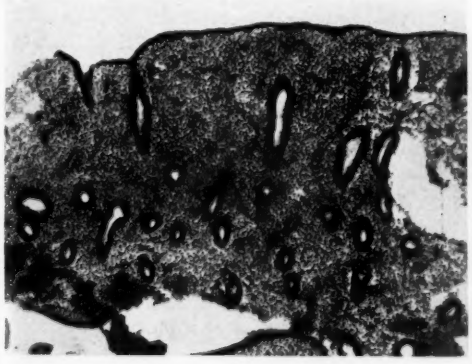

Fig. 3. Early proliferative phase. Specimen obtained on the sixth day in the normal cycle in a young patient. Straight tubular glands, three per low power field, mitosis active. Gross measurement 1 millimeter. Stained with hematoxylin and eosin (×50).

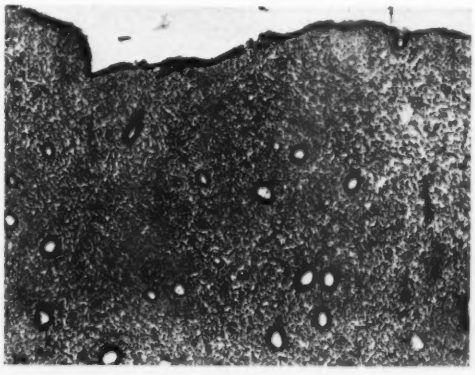

Fig. 4. Early proliferative phase (cross section). Specimen obtained on fifth day of cycle, illustrating early proliferative phase when glands are seen on the cross section only. Other features similar to early phase. Stained with hematoxylin and eosin (×35).

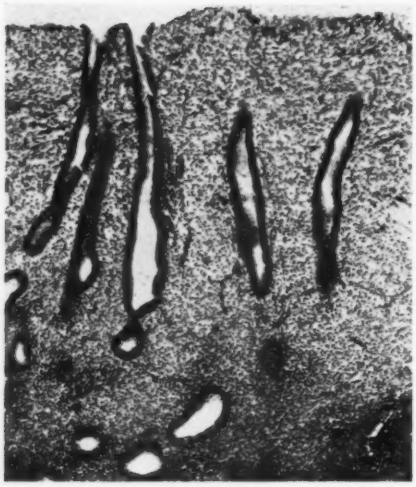

Fig. 5. Late proliferative phase. Specimen obtained from a patient with a normal cycle, on the ninth day. Straight tubular glands somewhat dilated as compared with early phase. They average 6 per low power field; gross measurement 2 millimeters. Hematoxylin and eosin (×40).

process of cyclic regeneration prevents such an occurrence, hence, from these studies, it becomes obvious that the earlier descriptions of the endometrium as a tissue of fixed, stereotyped, histological structure were entirely erroneous. During this phase proliferation continues rapidly, new glands form from the surface epithelium, and the stromal cells in a given field nearly double in number. By the end of the second week of this late proliferative phase, the average number of glands per low power field is six to seven; these glands are somewhat dilated, but they remain of the straight tubular type and show little if any evidence of a differentiative change. The endometrium grossly is from 2 to 2.5 millimeters thick (Fig. 5). It is worthy of note that this phase corresponds roughly to the life of the graafian follicle, and it would therefore seem that the graafian follicle has to do with the proliferative phase of this process of regeneration. At the end of this phase, proliferation is on the decline; however, evidence of slight proliferation is seen in the next phase, another striking correspondence to the failing follicular activity.

Early differentiative phase (fifteenth to twenty-first day). This has also been called the interval phase by Novak; by others, it has been called the phase of premenstrual endometrium. We would again propose descriptive terms, however, which would at once indicate the histological as well as the physiological state of the endometrium. One of the earliest indications of differentiation is the beginning convolution of the longitudinal glands. The epithelium, which up to this time has been of the proliferative type, that is, low columnar epithelium, changes to the columnar type and the nuclei approach the base of the cells. There is some evidence of proliferation, namely, an increase of cells in the stroma. By the end of the third week of this early differentiative phase, the endometrium grossly is about 3 to 3.5 millimeters thick; the number of glands, however, is not increased as compared with the number in the proliferative phase, the average number remaining six to seven per low power field. These glands are dilated and there is only an apparent increase in the stromal cells, which has been called continued proliferation by most workers (Fig. 6). It is of note that this phase is identical with that of early activity of the corpus luteum and failing graafian follicle, which histologically is expressed here as decreasing evidence of proliferation and a rather sudden onset of all of the histological evidence of differentiation. It is with the early part of this phase that one

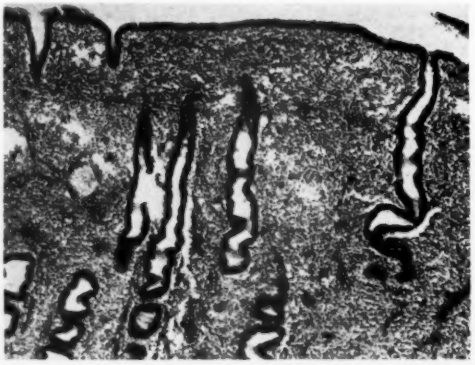

Fig. 6. Early differentiative phase. Specimen obtained by biopsy from patient with normal cycle, on the fifteenth day. Note beginning convolution of longitudinal glands. Six to seven glands per low power field; gross measurement 2.5 centimeters. Stained with hematoxylin and eosin (×35).

Fig. 7. Late differentiative phase. Specimen (biopsy) obtained on twenty-fourth day from a patient whose cycle was entirely normal. This is a typical late differentiative phase, that is, six to seven glands per low power field, glands twisted on longitudinal axes, and tall columnar epithelium. Gross measurement 4 millimeters. Stained with hematoxylin and eosin (×40).

may have difficulty in classifying endometrium, especially in fresh section. The fixed preparation, however, using the criteria, will supply the necessary information.

Late differentiative phase (*twenty-second to twenty-eighth day*). This may be termed the premenstrual phase, although such a term does not describe the true state of endometrium. In this phase differentiation is at its height and is characterized by certain changes (Fig. 7). The glands, which in the early differentiative phase disclosed beginning convolution, are now twisted on their longitudinal axes, producing the typical corkscrew glands, or, as Schroeder termed them, *saegefoermigedruesen*. When one views these glands in longitudinal section they look like a sectioned seashell with the typical saw-toothed appearance of the epithelium. This twisting, we believe, is a characteristic of differentiation whereby the surface area is increased. The epithelium is distinctly columnar and the

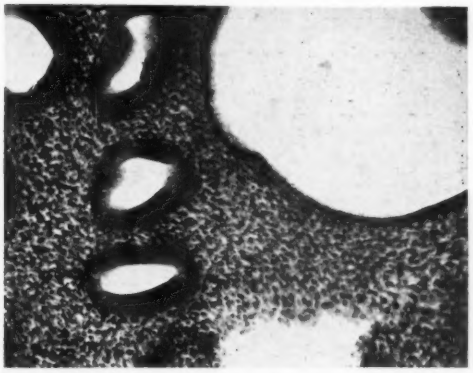

Fig. 8. Cystic endometrium. Specimen obtained on the twenty-third day of a normal cycle. Typical late differentiative phase with cystic areas. Patient aged 46 years. Note height of functioning epithelium as compared with epithelium of cyst. Menopausal symptoms present. Stained with hematoxylin and eosin (×78).

functional activity is marked. There is a definite cytoplasmic increase, with a decreased nucleo-cytoplasmic ratio, a distinct differentiative phenomenon. Glycogenic material is increased in these tall cells and the nuclei are displaced nearly to the basement membrane. The lumina of the glands at this phase are increased in size, and hence, the stroma by mere mechanical pressure is apparently increased. By the end of this phase the endometrium grossly is approximately 4 millimeters thick, yet the average number of glands per low power field remains the same as at the end of the proliferative phase.

Just prior to menses there is premenstrual engorgement of the stroma and infiltration with wandering cells, together with some edema near the surface. This engorgement is a generalized vascular reaction in the arterioles in definite contradistinction to the focal hemorrhagic areas seen in specimens obtained at dilatation and curettage.

HYPERTROPHIED ENDOMETRIUM

This term has been used rather loosely, and, according to available data, it conveys little knowledge concerning the histology of the endometrium. The term was first introduced in the literature by Cullen (1900), at the suggestion of Dr. William H. Welch. Gardner and Novak (1909), Warner (1915), and Novak

TABLE I.—ANALYSIS OF 188 CASES IN WHICH A DIAGNOSIS OF HYPERTROPHIED ENDOMETRIUM WAS MADE

Phase	Days	Cases	Glands per low power field	Average thickness of endometrium, mm.
Early proliferation	1 to 7	24	3	1+
Late proliferation	8 to 14	34	6	2.5
Early differentiation	15 to 21	34	6 to 7	3.5
Late differentiation	22 to 28	60	6 to 7	4.0
Menopausal and postmenopausal		36		
Total		188		

(1921, 21) later popularized the term; in fact, Novak is greatly responsible for its widespread use. Novak (20), however, recognizing the difference between the so called hypertrophy and what he thought to be another entity, introduced the term hyperplasia. The microscopic picture of hyperplasia he described as small tubular glands associated with dilated areas lined with flat epithelium. These cyst-like areas have been said to be pathognomonic of hypertrophy. The significance of this statement will be considered later; suffice to say, however, the observations made here will not support this theory. In a study of 159 cases in which the endometrium was removed before the fourteenth day of the menstrual cycle, the diagnosis of hypertrophy was made only once by Novak. This is but further proof that the differentiative phase in the menstrual cycle has been repeatedly confused with hypertrophy. In this same series of cases, the diagnosis of hyperplasia was made several times by Novak but always in cases in which there was a climacteric history and in which the patients were more than 40 years of age.

Others, as well as Novak, said that there could be no bleeding without hyperplasia; however, time has tempered to a great extent this view. Excellent contributions by Anspach (1934), Suggs (1934), Hartman, as well as others, offer ample theoretical evidence to refute any relationship between the so called hypertrophy or hyperplasia and the clinical intensity of menstruation.

To clarify this confusion, we would like at this point to define the terms "hypertrophy" and "hyperplasia," from standard medical dictionaries. "Hypertrophy" may be defined as the morbid enlargement or overgrowth of an organ or part. Likewise, "hyperplasia" may be defined as the abnormal multiplication or increase in the number of tissue elements. It is obvious, therefore, that the use of these terms as descriptive pathological terms is entirely misleading and without substantive histological support.

As a test of the significance of such a diagnosis, a careful study was made of sections prepared from 188 specimens from which a diagnosis of hypertrophied endometrium had been made in this department. The clinical history was not consulted until the phase of the cycle had been determined according to the criteria proposed in this report. A careful analysis of the data obtained reveals no justification for such a diagnosis without qualification. Reference to Table I will indicate the experimental basis for the facts which have been stated.

Interestingly enough, the diagnosis was made more frequently in the late phases of the menstrual cycle than it was in the early phase; however, the diagnosis not infrequently has been made from specimens measuring 1 millimeter in thickness, specimens containing three glands per low power field, and specimens presenting all the characteristics of early proliferative endometrium. These do not represent selected specimens; they were surgical specimens, routinely diagnosed as hypertrophied endometrium in 188 consecutive cases.

CYSTIC ENDOMETRIUM

Reference has been made to the appearance of cystic areas occurring in endometrial specimens, which are often diagnosed as hypertrophied endometrium. This is an error caused by faulty interpretation of these cystic areas, which accompany clinical ovarian failure or

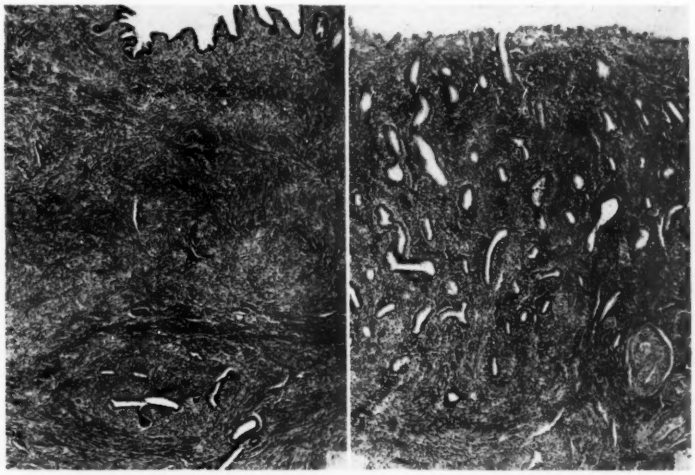

Fig. 9. Left, Subbasal glands. Specimen showing subbasal glands 1 millimeter below the surface epithelium in a fetus, 30 weeks of age. Stained with hematoxylin and eosin (×35). Right, Subbasal glands (so called internal adenomyosis). Specimen obtained from menstruating uterus of a girl 15 years of age, showing normal cycle. Note subbasal glands which are said to occur only at menopause. Stained with hematoxylin and eosin (×35).

the approaching menopause rather than an associated hyperplasia.

This picture has been described by some as the "swiss-cheese" endometrium; however, we propose the term "cystic endometrium" and would point out its significance. During this study, cystic areas have been observed in approximately sixty endometrial specimens, none of which could be classed as hypertrophied endometrium. In all cases there has been clinical evidence of ovarian failure or approaching menopause. The occurrence of these areas in the endometrium of a patient, even with a normal cycle, heralds the approaching menopausal age with such consistency that one usually can predict without the clinical data the approximate age of the individual. Shaw, Graves(14), Cannon, and more recently, Duhig, have postulated, without experimental support, that this "cystic hyperplasia" is often associated with ovarian (follicular) dysfunction. Likewise, Burch, in a study of so called endometrial hyperplasia, has reiterated the fact that corpora lutea are not present in the ovaries of patients who come to operation suffering with "cystic glandular hyperplasia and in fact the ovaries are generally cystic." This is a pertinent observation. Since the work here presents substantive histological evidence that the secretion of the corpus luteum has to do with differentiation (previously called pregravid endometrium), and since this phase is admittedly the picture called by most workers hypertrophy, it becomes obvious that such a diagnosis is incorrect. Rather the histology is one of arrested regenerative phenomenon in an early phase and the cysts result from failure of the ovarian tissue. Cystic areas associated with arrestment of the regenerative process will be discussed later. Figure 8 illustrates a typical example of the cystic endometrium of a patient whose cycle is still normal, but who without question is approaching the menopausal age. Cystic areas have been observed also in young patients whose endometrium was not hypertrophied according to gross measurements or microscopic examination but who presented clinical syndromes of ovarian failure. The practical significance of this observation is obvious as regards therapy.

SUBBASAL GLANDS

Attention is again called to the small glands for which we have proposed the term "sub-

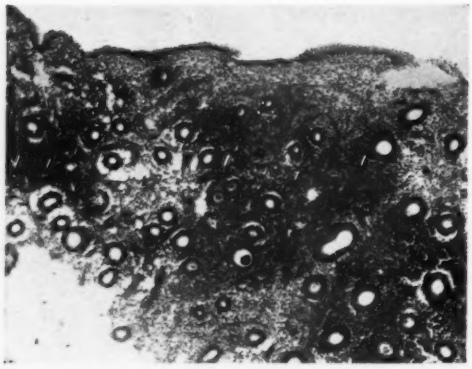

Fig. 10. Persistent early proliferative phase. Specimen shows typical early proliferative phase in a patient aged 27 years. Oligomenorrhea (periods varied from 2 to 3 months apart). Section identical with those previously diagnosed hyperplasia. Stained with hematoxylin and eosin (×35).

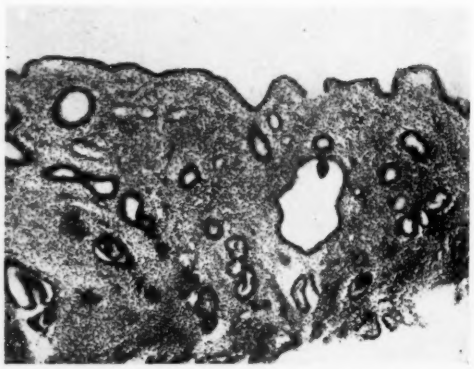

Fig. 11. Persistent early proliferative phase in a cystic endometrium. A typical menopausal endometrium. Patient, aged 41 years, gave a history of amenorrhea of 6 months' duration. Note cystic area, condition previously called hyperplasia. Gross measurement 2 millimeters. Stained with hematoxylin and eosin (×40).

basal glands." It has been stated by Shaw, Taylor, Frankl, and Adler, Jeffcoate and Potter, as well as by Graves (15), that the presence of these glands is pathognomonic of the menopausal age, and some observers even say also pathognomonic of hyperplasia. These workers have claimed that this process is histologically identical with external adenomyosis, or the Sampson tumor. These same observers, however, are unable to present clinical features characteristic of this process and frankly admit that the glandular structures of internal adenomyosis do not participate in the menstrual cycle. It is inconceivable to us that two identical histological tissues could react in an exactly opposite manner to the same endocrine stimulus. These structures have been observed during all phases of the menstrual cycle although we have never seen them participate in the histological changes in any way. Further, it is of interest that Frankl's series of 30 cases, revealed according to his results the lowest age incidence at 41 years for the presence of these glands, in support of his theory of a preclimacteric disease. We have observed and photographed these glands (Fig. 9) at all ages of the endometrium, ranging from an intra-uterine fetus, 30 weeks of age, to 20 years past the menopause. We are, therefore, unable to subscribe to such a theory. It seems reasonable to say from these observations that these tissues are definitive structures (perhaps examples of heterotopic tissue) possessing the possibility of activity yet uninfluenced as a rule by the endocrine stimuli.

PERSISTENT PHASES OF THE MENSTRUAL CYCLE

The practical application of this classification which has been established for the normal, has been attempted in the study of various types of menstrual disturbances. The histological picture presented by both the bleeding and non-bleeding endometria, may be classified and interpreted according to the basic principles revealed by the previous studies. Endometrial specimens were obtained from patients who presented themselves with symptoms of amenorrhea for biopsy and for dilatation and curettage. By these methods, specimens have been obtained of endometria arrested in every phase of the cycle. According to the histological picture, these states have been designated by us as "*persistent phases of the cycle.*" Partial or complete ovarian failure expresses itself with great preciseness in the histology of the regenerating endometrium. The practical value of knowledge gained by such a study need only be mentioned.

Arrestment of the process of regeneration in the proliferative phase, either early or late, is indicative of ovarian failure, at least to the degree of deficiency of corpus luteum and

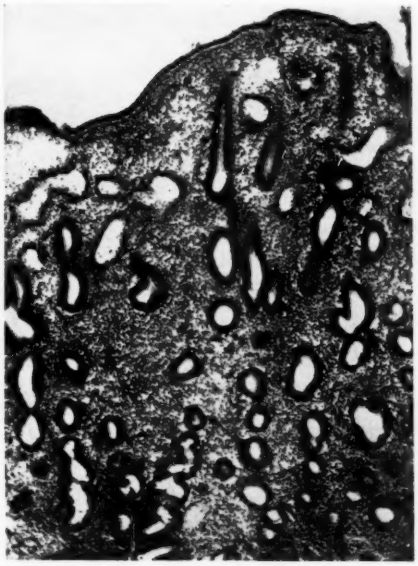

Fig. 12. Persistent late proliferative phase. Specimen removed from patient aged 23 years. Last menstrual period 3 months before her admission to hospital; a regular irregularity with occasional molimina. Typical late proliferation; six glands per low power field; straight dilated tubules on cross section. Stained with hematoxylin and eosin (×40).

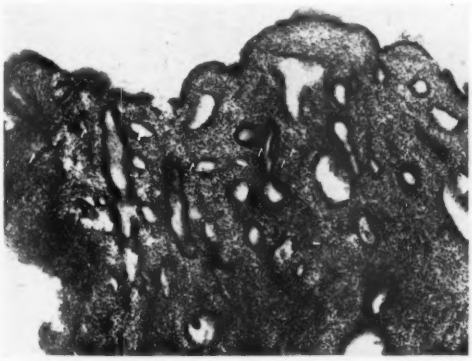

Fig. 13. Persistent early differentiative phase. Specimen removed from a patient aged 30 years. Menstrual interval 3 to 6 months for past 2 years. Specimen obtained 3½ months after last menstrual period. Note beginning convolution of longitudinal glands and six glands per low power field. Other features of early differentiation. Stained with hematoxylin and eosin (×35).

probably lack of folliculin, since it has been shown that the corpus luteum has to do with differentiation.

Persistent early proliferative phase. Figure 10 illustrates a typical early proliferative phase. Such an endometrium reveals small tubular glands on cross section and in every way is identical with a normal endometrium in the early proliferative phase. However, this endometrium was removed from an individual, 27 years of age, who presented herself with symptoms of amenorrhea. The menstrual periods varied from 2 to 3 months apart, and there was other clinical evidence of ovarian failure, as well as sterility. Figure 11 is a photomicrograph of the endometrium of a patient who presented herself with symptoms of amenorrhea which had had its onset 6 months before her admission to the clinic. She had had a few "hot flashes" before the onset but denied that she had had any since that time. This patient, however, was approaching the menopause, as is attested by the occurrence of cysts in the endometrium. These typical cystic areas occur in the early proliferative phase. This finding we have said to be indicative of ovarian failure. Despite the occasional appearance of these cystic areas in the endometrium of a young woman, this finding is typical of physiological senile change. This type of endometrium is almost identical with that which previously has been called hyperplastic. In describing this type of endometrium, Gardner (1915) was the first to express the opinion that the glands are occasionally mistaken for the normal postmenstrual type of endometrium. He further said that this type of endometrium was associated with cyst-like areas. It is obvious, therefore, that he has in substance described an early proliferative phase in a cystic endometrium. Further evidence in support of the interpretation we have placed on this type of endometrium is attested by the following statements. Novak (1920, 24), in a description of this so called hyperplasia, observed that no matter at what period of the cycle the endometrium was curetted, the picture remained the same; that is, there were small, irregular, straight tubules, associated with cystic areas. He was of the opinion, in contrast to previous statements, that the influence of the corpus luteum was in

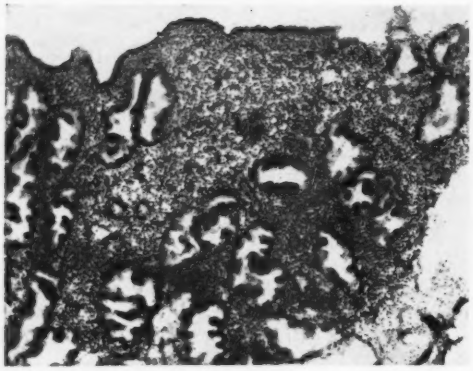

Fig. 14. Persistent late differentiative phase. Specimen removed from patient aged 26 years. Regular irregularity with menstrual interval from 2 to 4 months for past 3 years. Last menstrual period 3 months before her admission to hospital. Specimen identical with late differentiative phase (see Fig. 7). Stained with hematoxylin and eosin (×35).

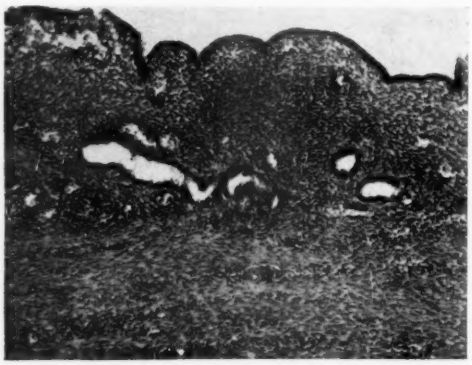

Fig. 15. True atrophic endometrium. Specimen of uterus removed several years after removal of both ovaries. Endometrium similar to prepubescent type. Single layer of epithelium overlies loose stroma with few non-functioning basal and subbasal glands. Stained with hematoxylin and eosin (×60).

abeyance. Fluhmann (1931), in a very painstaking study, observed that the ovaries of patients with hyperplasia (usually just before and during the menopause) characteristically showed ripening follicles with complete absence of mature corpora lutea. Thus, there was a complete absence of the influence of progestin. It is logical, therefore, to reason from these observations that the several observers have described a state of the endometrium which is the result of ovarian failure to the degree of absence of the hormone of the corpus luteum. Since these observations rather conclusively demonstrate that differentiation of the endometrium is dependent on this hormone, it becomes obvious that the regenerative process will proceed only to that point controlled by the follicular hormone and will proceed no further when activity of the corpus luteum is absent. This results in a persistent proliferative phase and not in hyperplasia. The endometrium usually does not measure, in the gross, more than 1 to 2 millimeters at any time. There are three longitudinal glands per low power field, and the epithelium is of the low proliferative type. All of these features are further evidence that this condition is not a hyperplasia. In this study, 16 specimens of this type of endometrium have been collected from patients who presented typical menopausal symptoms and from patients who had symptoms of amenorrhea.

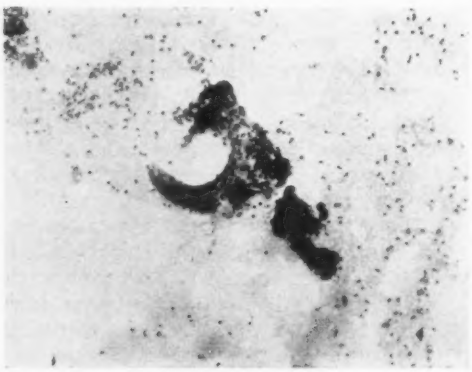

Fig. 16. True atrophic endometrium. Small islands of epithelial cells in a loose stroma. Stained with hematoxylin and eosin (×80).

Persistent late proliferative phase. Similar to patients seen in the first phase of the menstrual cycle, there are cases in which the regenerative process progresses to a point further in the cycle and becomes arrested at this point. Figure 12 is a photomicrograph of a section of endometrium removed from a patient aged 23 years, who had oligomenorrhea (in this case menstruation occurred 3 to 6 months with occasional molimina). The last menstrual period had occurred 3 months before her admission to the clinic. This section illustrates a

typical late proliferative phase of the menstrual cycle; that is, there are approximately six longitudinal glands per low power field. These glands are somewhat dilated but straight. The histological appearance of this section presents nothing which would indicate deviation from the normal; however, from the history, it must be called a persistent proliferative endometrium. This indicates a deficiency of corpus luteum, that is, the stimulus for regeneration is present in sufficient quantity to bring about complete proliferation, but differentiation is lacking and hence menstruation does not occur. From a therapeutic standpoint, this observation is of great importance in supplying the necessary deficient hormones.

Persistent early differentiative phase. Examples of endometrium have been encountered which are definitely in a persistent differentiative phase. Figure 13 illustrates the endometrium of a young woman who had had irregular menstrual periods for 2 years. There had been an interval of 3 to 6 months and an occasional molimen. This specimen is typical of the early differentiative phase. There are six glands to the low power field, some increase in the height of the epithelium, and evidence of beginning convolution of the glands, all of which are characteristic features of early differentiation. The condition illustrated by this section should respond to ovarian stimulation, which in turn should bring about differentiation, thus completing the process of regeneration.

Persistent late differentiative phase. Cases have been seen in which histological study revealed a very slight ovarian deficiency, or at least a lack, of the factor which would initiate the menses. Figure 14 is a section of endometrium removed from a young woman whose menstrual periods had occurred at intervals of 3 to 4 months; however, there always had been a definite molimen at the expected date. This section shows a typical late differentiative phase of the menstrual cycle. There are six or seven longitudinal glands per low power field, the glands are twisted on the longitudinal axes, and the epithelium is of the tall columnar type. The nuclei are displaced toward the base of the cell. The gross measurement is between 3.5 and 4 millimeters. It is, therefore, reasonable to say that this is an example of a persistent late differentiative phase. In light of the foregoing observations, it may be assumed that slight ovarian stimulation should be sufficient to bring about a readjustment in the cyclic regenerative changes in this type of endometrium.

ATROPHIC ENDOMETRIUM

The term "atrophic endometrium," like the term "hypertrophic endometrium," has been used rather loosely. It has been applied to menopausal as well as postmenopausal endometrium; however, observations made in this study would indicate that true atrophic endometrium would be more applicable to that type of endometrium in which complete and rather sudden ovarian failure has occurred. Following sudden failure (either spontaneous or artifical failure, such as occurs after surgical removal of the ovarian tissue), there occurs a lack of both the follicular hormone and the hormone of the corpus luteum, which are the stimuli to endometrial regeneration, and hence true atrophy occurs. The gradual failure of the ovaries incident to the climacteric usually is associated with cystic formation, as stated before. However, sudden withdrawal or failure of ovarian tissue is accompanied by a true atrophy (Fig. 15). This figure illustrates a section of uterus removed from a patient whose ovaries had been removed several years before. The remaining endometrium consists of a single layer of epithelium overlying a loose stroma. This is typical of true atrophic endometrium. In this type of case biopsy or even vigorous curettage will secure only small amounts of endometrial tissue. However, a diagnosis can be made from the presence of small islands of epithelial cells which usually are situated in the loose stroma described previously. Figure 16 illustrates a true atrophic endometrium removed from a patient aged 29 years, who had had a sudden onset of amenorrhea 12 months before her admission to the hospital. From this histological picture, it may be concluded that therapy, regardless of type or duration, probably will do little to restore this endometrium. Nine such cases have been studied; however, they serve to indicate the difference between true atrophic and the

TABLE II.—RESULTS ACCORDING TO CLASSIFICATION PROPOSED IN THIS STUDY*

	Source of material		
	Hysterectomy	Dilatation and curettage	Biopsy†
Phases of cycle (normal)			
Early proliferative phase	11	25	4
Late proliferative phase	16	16	9
Early differentiative phase	14	29	13
Late differentiative phase	20	17	9
Persistent phases of cycle			
Early proliferative phase	0	9	7
Late proliferative phase	0	0	1
Early differentiative phase	0	2	1
Late differentiative phase	0	1	2
Cystic endometrium	24	30	6
True atrophic endometrium	1	2	6
Subbasal glands	7	3	1
Postmenopausal	4	5	0
Menstruating ("tissue loss")	4	1	0
Total	101	140	52

*Diagnosed hypertrophied endometrium, 188.
†Note that 21 per cent (11) of the specimens in this group fall in the classification of persistent phases of the menstrual cycle. This represents the usefulness of the biopsy together with this histological study in the diagnosis of endometria of patients with amenorrhea which results from endocrine disturbances. A few of these cases were studied also by the dilatation and curettage.

menopausal type of endometrium, which is usually a persistent early proliferative phase in a cystic type of endometrium.

ANALYSIS OF STUDY

The specific observations have been considered previously in the separate sections. An effort has been made to consider briefly the different phases of the regenerating human tissue heretofore mentioned. It becomes clear, after consideration of the results following tissue loss, that they can be explained in terms of a few general basic principles at work in the regenerative process. The significance of these observations becomes obvious when one considers them in the light of an analysis of the regenerative phenomena. The different phases have been cited previously. Following injury, or spontaneous tissue loss, approximately 48 hours are taken up with migration and reorganization of cells and this is the vital period in the whole process of recovery or regeneration. Following this, proliferation goes on actively and is associated with frequent cellular division and, at times, with active mitosis. This mitotic activity usually progresses rapidly until the tenth day and declines at the beginning of the third week. It is interesting that Westphalen, as early as 1896, noticed that mitosis in the endometrium was most marked from the seventh to the eighteenth day of the cycle. However, he was unable to explain this observation. Likewise, Novak noted that mitosis never occurred during the loss of tissue, an observation which he also was unable to explain. In light of our present knowledge of regeneration of tissue, it becomes obvious that Novak was observing the phase of cellular migration which lead him to postulate that cellular division took place by amitosis. Likewise, Westphalen was observing the period of active proliferation which is practically completed by the eighteenth day following which the process of regeneration is one of differentiation, and hence very little mitotic activity is to be observed.

Regarding the rôle played by the hormone of the corpus luteum (progestin) in the differentiation of the endometrium, one needs only to say at this time that comparative histological studies alone indicate that the rôle is very definite. A demonstration of the potency of the follicular hormone by such investigators as Allen, Doisy, and their collaborators was of great aid in these studies; however, too much emphasis was placed on this hormone. Hence, the discovery of the principle of the corpus luteum was delayed. At present, ample experimental proof exists to establish clearly the fact that ovulation occurs in the human being and that this usually occurs at the middle of the cycle (fourteenth to fifteenth day). This is followed by activity of the hormone of the corpus luteum and by decreasing activity of folliculin.

A consideration of the experimental evidence, however, does not come within the scope of this paper. It may be noted, notwithstanding, that ample histological evidence has been gathered from endometrial specimens which, when correlated with accurate clinical histories, establishes the fact that differentiation is definitely under the

control of the corpus luteum. The removal of this stimulus therefore results not in hyperplasia or hypertrophy, but in an arrestment of the regenerative process in its early phase.

Table II illustrates a complete analysis of the material studied.

CONCLUSIONS

1. A careful histological and clinical study of 293 specimens of endometrial tissue obtained in various phases of the menstrual cycle has been presented.

2. Some new conceptions of the basic principles at work in the regeneration of human tissue have been presented, with experimental evidence to confirm these conceptions.

3. Careful analysis of the results of this study reveals the fact that one is not justified in making a diagnosis of hypertrophied or hyperplastic endometrium without qualification.

4. The practical significance of cystic areas occurring in the endometrium has been indicated, with histological evidence that this is a phenomenon indicative of physiological senility.

5. Experimental evidence we believe, presents incontrovertible evidence that the subbasal glandular tissue is not a disease process pathognomonic of the preclimacteric state but rather a definitive tissue possessing potential possibilities of activity under certain conditions.

6. The value of a histological study and classification of endometrial tissue arrested in its process of regeneration, because of a deficiency of the humoral stimuli which control this process, has been thoroughly demonstrated ("persistent phases").

7. The practical value of a differentiation between true atrophic and menopausal types of endometria has been indicated.

8. As a result of this study, the following classification is used in the diagnosis of endometria in Section B on Surgical Pathology at The Mayo Clinic.

1. Phases of cycle (normal)
   - Early proliferative phase
   - Late proliferative phase
   - Early differentiative phase
   - Late differentiative phase
2. Persistent phases of cycle
   - Early proliferative phase
   - Late proliferative phase
   - Early differentiative phase
   - Late differentiative phase
3. Cystic endometrium
4. Postmenopausal endometrium
5. True atrophic endometrium
6. Subbasal glands
7. Menstruating ("tissue loss")

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AN EXPERIMENTAL STUDY OF THE EFFECTS OF CONSTRICTION OF THE GREAT VESSELS OF THE HEART[1]

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"Dii talem avertite casum"—Virgil

THE serious results of pulmonary embolism are well recognized and in the last few years have received more and more attention. The apparent increase in frequency may be due to more accurate diagnosis, although Nurnburger, after an analysis of statistics, concludes that in recent years there has been an actual increase in thrombosis and embolism, but makes the reservation that this increase is not so great that the accidental can be excluded.

Pulmonary embolism is a clinical entity usually considered as a condition to be treated prophylactically, rather than surgically. Eichelter says that only every fourth to sixth case is suitable for the Trendelenburg operation, but Nyström believes that because of our limited experience we cannot say where the line for operability has to be drawn with regard to the length of time the pulmonary artery can remain obstructed without proving fatal.

Scheidegger, in his report of 20,779 operations, noted a general occurrence of fatal pulmonary embolism in 0.69 per cent of cases with a fatality of 4.04 per cent from this cause in the exploratory laparotomies in this group. Snell, of the Mayo clinic, says that pulmonary embolism is responsible for 8 to 10 per cent of all postoperative deaths. Any malady of such frequent occurrence and high fatality must surely cause us to search for efficient restorative measures. To treat the condition prophylactically only is to disregard a large group of cases which may well become amenable to active surgical therapy. It was with the hope of contributing in some measure to a method of giving more efficient restorative treatment that this experimental work was carried on.

HISTORICAL REVIEW

Virchow, in 1846, was the first clearly to describe pulmonary embolism and to correlate the relationship between thrombosis and pulmonary embolism. He first grouped the obstruction of the pulmonary artery under four general headings. In the same year he published additional work in which he described the mechanism of occlusion of the "Lungenarterie," giving the results of his experiments on animals. He injected foreign materials and observed the secondary disturbances resulting from the obstruction of the pulmonary vessels. He reported the autopsy findings. Church, in 1892, writes concerning Van Sweiten:

> As far back as 140 years ago Van Sweiten recognized that clots occurred in the vessels during the puerperium and wrote gravely of their prognosis. He even performed experiments on animals, injecting acids into the veins, and bringing about phenomena in some cases identical with what we observe in phlegmasia dolens, and in others, the severe and sudden symptoms which we know to characterize thrombosis and embolism of the right side of the heart and pulmonary arteries.

The first interest in pulmonary embolism as a surgical problem was taken by Trendelenburg after he had noticed that patients stricken with a pulmonary embolus did not die suddenly but might live 15 minutes or longer. His interest in this subject led him to carry on experiments to determine the possibilities of removal of an embolus lodged in the pulmonary artery. He reported his work before the Versammlung deutscher naturforscher und Aerzte in Dresden in 1907.

Trendelenburg's first work was carried out on cadavers, through a great flap on the left thorax. The pericardium was exposed by a wide pleural incision and was opened widely. A 50 cubic centimeter syringe was prepared with a two-way valve which was fitted to a cannula through an obturator with a lateral clamp. Two sutures were placed about 1 centimeter apart in the wall of the conus arteriosus at a point 1 to 2 centimeters to the

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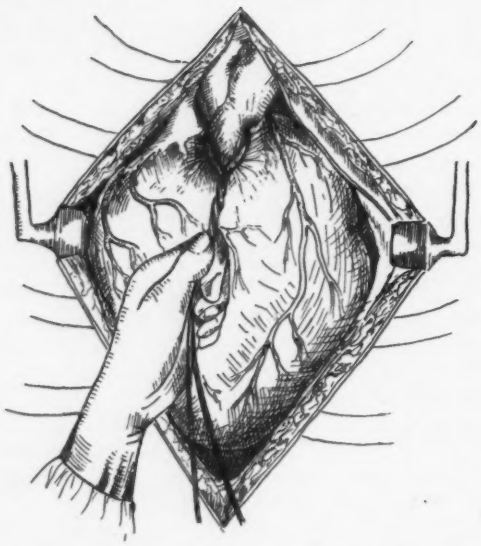

Fig. 1. This illustration shows the constrictor tightened about the pulmonary artery and aorta with the enormous distention of the heart which results. (After the method of Trendelenburg.)

right of the sulcus longitudinalis anterior and about 4 centimeters away from the semilunar valve. A puncture wound was made in the wall between the two sutures, with a pointed knife and the wound dilated about ¾ centimeter. The cannula was inserted through the wound and into the conus arteriosus and the opening of the pulmonary artery. The loose embolus was then sucked out into the syringe and forced out through a lateral connection at the end of which was a small wire basket to catch the embolus.

With the embolus floating in the vessel in water in the cadaver, the embolus could be caught with great agility when the heart was held. In experimental animals this was more difficult. Hirudin in salt solution was used in the beginning of the operation to facilitate the passage of blood in the apparatus, no comment, however, being made as to its effectiveness.

At the same time he reported a trial of this operation on a 62 year old woman with a carcinoma of the colon. At operation a dense adhesion was found between the pericardium and the heart so that the heart could not be exposed. In trying to free the adhesions a profuse hemorrhage occurred and death followed. After death, a 10 centimeter long, finger sized embolus was found in the left pleural space.

Laewen and Sievers, two of Trendelenburg's students, took up the experimental side of this problem, and in 1908 reported their results. They concluded that rabbits withstood complete interruption of the circulation for 1 minute without irreparable damage, but with compression longer than 2½ minutes the great majority of the animals succumbed. By the use of artificial oxygen respiration, heart massage, and adrenalin they were able to get a return of heart function after constriction of the pulmonary artery and aorta for as long as 7 or 8 minutes but they found irreparable damages in the brain and were unable to obtain function from the respiratory center. A partial compression for 10 minutes preliminary to constriction had no apparent effect.

From later experiments by Trendelenburg (35), it was concluded that it was much better to incise the pulmonary artery and remove the embolus than to attempt removal by suction. In 1908, he recommended the use of special forceps for removal of the embolus and a special artery clamp for closure of the vessel.

The operative procedure which was adopted and recommended by Trendelenburg (35) was to make a T-shaped incision above the left border of the sternum and laterally over the lower border of the second rib. The second rib was resected thus immediately exposing the pleura, the costal cartilage being sectioned close to the sternum. He advised the ligation of the internal mammary artery before proceeding farther. By incision in the pleura, the pericardium was exposed and then widely opened. The Trendelenburg sound was then passed around the pulmonary artery and aorta through the transverse sinus of the heart and the rubber tube attached by means of a clip after which the tube was drawn into place. The tube was then used for a constricture of the great vessels while the pulmonary artery was incised, and the embolus, located in the

pulmonary artery proper or in either the right or left branches, was extracted.

From the time Trendelenburg reported his first attempt at removal of a pulmonary embolus in the human in 1908 until 1923 there were, according to Eichelter, 20 failures with the original Trendelenburg method (Capelle, Krueger, Kuettner, Laewen, Ranzi, Rehn, Sauerbruch, H. H. Schmid, Schumacher, Sievers, and Trendelenburg) before the first permanent cure was reported. These results served to show the possibilities of the operation and the technical difficulties. A few cases survived the operation for longer periods but a successful outcome had not yet been reported. Sievers had one patient survive embolectomy for 15 hours and Trendelenburg (36) had one patient live for 37 hours after operation who died of a hemorrhage from the internal mammary artery. Both of these patients were treated in 1908. Schumacher, in Sauerbruch's clinic, had a case in which patient lived 50 hours and Krueger had a patient who lived 5 days after operation to die of pneumonia. It remained for Kirschner, one of Trendelenburg's pupils, to report the first successful case in 1924.

Kirschner used the Trendelenburg technique and was able to report the only successful case to the present date in which this method was used. A. W. Meyer (21) in 1926 operated upon a patient using the original technique but he was unable to save the patient. After dissections and experiments he pointed out a new technique which was designed to overcome some of the difficulties in the older method. Meyer (21) pointed out three factors in Trendelenburg's method which served to hinder or defeat the operation and which he proposed to avoid. These factors are as follows:

> (1) The opening of the pleura implies the infliction of a stupendous shock upon the already injured heart; (2) the Trendelenburg sounds and clip must be improved . . . ; and (3) the strangulation period of 45 seconds caused by the introduction of the rubber tube around the great vessels puts too much strain upon the already laboring and dilated heart (a sound heart may be able to withstand this period) and this period of strangulation evidently induces a paralysis of the respiratory centers which becomes irreparable.

The shock added because of a pneumothorax Meyer forestalled by an extrapleural approach which was secured by a wider exposure and pleural reflection in the region of the "triangle of safety." He had a sound made which was smaller and a pulmonary artery clip made shorter and narrower and covered with gauze instead of rubber. The strangulation time of 45 seconds was cut down by digital compression of the slit in the pulmonary artery after shorter intervals of work.

The technique of the A. W. Meyer modification of Trendelenburg's operation I will give in detail as he has given it:

> Since one is operating upon the dying, the operation is almost bloodless. The second and third rib must be laid bare with swift, large strokes of the scalpel. These ribs must be freed from periosteum and pleura quickly but very cautiously. The operator now gently pushes away the mammary artery and the pleura. The forefinger of the right hand is inserted to enter by way of the lower medial angle partly under the breast bone and the operator's hand gently feels its way under the insertion of the fourth rib. The pericardium, shining white and partly covered with fat, is plainly in view. Incision with the knife is followed by a flood of pericardial fluid. The pericardial cavity is laid open by introducing cautiously and energetically both forefingers which are spread out. Thus the pleura is pushed still farther out of place, and the pericardial cavity can be opened to a surprising extent.
>
> The next step is the insertion of my modified Trendelenburg sound and the placing of the rubber tube around the artery. On the cadaver one may be in doubt as to which is aorta and which is pulmonary artery. In the living, or rather the dying, body there can be no doubt the aorta lies hidden and the pulmonary artery, swollen and pulseless, is at once visible. The Trendelenburg rubber tube is useful, as we shall presently see, not so much in strangling the vessels as in bringing the large vessels out of the depths, which may be considerable in the case of fat patients, so that they can be better observed. The pulmonary artery is incised, the tube being held lower. A quantity of blackish blood rushes out. Emboli, which may come from the heart, are also flooded out. Greater tension is now applied to the rubber tube. Forceps are placed in the right pulmonary branch, and the Trendelenburg embolus forceps is now almost horizontal. If the right branch is found to be free of emboli, a triple investigation suffices, then the incision in the pulmonary artery is grasped with the thumb and forefinger of the left hand and is pressed together. The tube at once becomes absolutely relaxed. The blood is allowed to flow through the pulmonary artery for a few seconds, when, for the first time, the tube is fairly energeti-

cally tightened, and an almost vertical, triple penetration is made into the left branch. The heart is then relieved by means of renewed digital compression of the slit in the pulmonary artery, with the tube quite relaxed. Now for the second time, the tube is forcefully tightened. The left hand grasps the cranial end of the slit vessel with a pincers covered with gauze, and holds it aloft; the right hand attaches the arterial clip on the side; the tube is released. Any tightening of the tube naturally renders the work of the heart more laborious and thus injures it. The assistant must be cautioned regarding this, and must be ready instantly to obey the command: "relax," "more relaxed," "somewhat tighter," as the case may be.

Those surgeons who have had patients survive the operations are shown in Table I.

TABLE I

Operator	Year	Time survived
Sievers	1908	15 hours
Trendelenburg	1908	37 hours
Krueger	1908	5 days
Schumacher (Sauerbruch-Kl.)	1914	50 hours
Kirschner	1924	Discharged cured
Meyer	1927	Discharged cured
Meyer	1927	26 days
Crafoord	1928	Discharged cured
Crafoord	1928	Discharged cured
Nyström	1928	30 hours
Nyström	1928	5 hours
Nyström	1928	Discharged cured
Meyer	1928	Discharged cured
Meyer	1928	Discharged cured
Nyström	1929	Discharged cured
Nyström	1929	3¼ hours
Marcus	1930	8 days
Meyer	1931	Discharged cured
Westerborn	1931	16 hours
Cutler	1933	Unreported

There have been a total of 51 surgeons, all but one of whom have worked in European clinics, who have performed the Trendelenburg operation a total of 132 times with 19 patients who have survived the operation and 9 who have been discharged cured.

The work of A. W. Meyer (with his modification of the Trendelenburg technique) gave a great impetus to the work in this field, so that 132 such operations have been reported to date, only 25 of which were done before the use of the Meyer modified technique. Eichelter lists 123 of these cases operated upon up to 1932, giving the surgeon and the results. Two additional cases have been reported by Griswold in this country and more recently Cutler and his associates have operated upon and reported 7 cases in which embolectomy has been performed. In 1932, Meyer (23) was able to report his fourth successful embolectomy out of 16 reported cases. Nyström has made contributions to this procedure by modification of the instruments used previously and construction of a triple suction apparatus. He modified the Trendelenburg sound of Meyer so that the tube when clipped into the sound would not slip out as some operators had experienced and he also has modified the pulmonary artery forceps. Nyström used his suction apparatus successfully in extraction of large clots which could not be readily removed with the forceps. Meyer (22) has recently used a clamp for holding the pulmonary artery that is angulated.

THE PROBLEM

In the present method of performing pulmonary embolectomy, the duration of constriction must be so brief as to be a serious handicap. The results of too prolonged constriction have been marked by deterioration in the vital centers of the brain. It has been rather generally accepted that these deteriorations in the vital centers are due to ischemia.

The original concept of this problem was based on the fact that when the arterial blood supply to a part is interrupted or seriously impaired, the viability of the affected part is greatly dependent upon the amount of blood which can be retained in the vascular bed. The first observation on this point was made by Sir George Makins, about 1913, from his experience in the Boer War when he noted the occurrence of a high percentage of gangrene when proximal ligation was done for arteriovenous aneurisms or aneurismal varices while quadruple ligation and excision of the aneurism was followed by good results. In 1913, W. A. Oppel reported marked improvement in 6 cases of senile gangrene in which he ligated the popliteal vein. In 1917, Van Kend after doing some animal experiments, recommended concomitant vein ligation to "retain the blood supply in longer contact with the tissues." McNealy noted circulatory improvement in threatened gangrene of the extremities following main vein ligation. Some of the patients showed only temporary im-

provement but there occurred a noticeable decrease in pain and paresthesias following operation.

It seemed a logical hypothesis that an interruption of the circulation by constriction of the great vessels of the heart on the venous side would cause a stasis in the vascular bed of the medullary and higher centers and thus protect the most vulnerable spot when the circulation is completely interrupted. Death of the respiratory center has been demonstrated in the case of embolectomy done by Nyström who was able to get a heart beat for 7 minutes following constriction, but respiration was never resumed. Ranzi had a similar case with the heart beat lasting for 40 minutes. Kirschner was able to get a heart beat for 2 hours with artificial respiration but with complete failure of the respiratory center.

The classical experiments of Laewen and Sievers have shown that the heart can withstand blocking of the circulation for longer periods than the brain and medullary centers. The example of the effect on the brain as shown by Kirschner's successful embolectomy may be cited. Following the operation the patient was alternately delirious and comatose for 4 days before regaining consciousness. In Crafoord's first successful case the patient was unconscious for several hours, was cold and clammy, and for 2 days lay in a shock-like condition with complete amaurosis and was at times delirious. On the third day her sight returned and no cerebral symptoms followed.

An attempt was made to demonstrate a prolonged viability and more rapid resumption of activity of the medullary centers by developing a practical method of constriction of the superior vena cava. By the retention of venous blood in the brain, which we know to have a prolonged nourishing effect, it was hoped a better circulatory balance could be maintained during the period of interruption of circulation.

It is interesting to note that venous blood returned from the brain has a much higher oxygen reserve than does venous blood returned from the limbs. The nourishing effect from maintaining venous blood in contact with the tissues would accordingly be most effective in the brain. Howell has gathered varying reports concerning the oxygen content in volumes per cent of blood in arterial and venous states but gives them as:

	Vols. per cent
Arterial blood	20.
Venous blood	12.
Venous blood from limbs (femoral)	6.34
Venous blood from brain (torcular)	13.49

In certain anemic persons the oxygen capacity of blood may be as low as 6 to 7 per cent, owing to the reduction in the amount of hemoglobin. If the tissues take up 5.5 volumes per cent of oxygen, the blood enters the veins in a state of almost complete oxygen unsaturation. The large oxygen reserve in normal blood available to the respiratory center if the blood may be kept in contact with it during the time of suspended animation, should have a beneficial influence on its viability.

One might question the explanation of the failure of the respiratory center on the basis of ischemia when one considers the fact that it is easy to demonstrate early dilatation of the right auricle following occlusion of the pulmonary artery. One would reasonably conclude that such evident dilatation of the right auricle would produce sufficient passive congestion in the superior vena cava to insure against early ischemic changes.

It is probably not inconsistent to suggest that this apparent discrepancy can be explained by assuming that the inferior vena cava will accommodate so large a proportion of the total blood volume without material increase in its intravascular pressure that the superior vena cava will not reach a state of sufficient pressure to insure widespread resistance to the rapid emptying of the minute arterial capillaries until irreparable damages to such centers as the respiratory center have resulted.

A further explanation of the inadequacy of the back pressure in the superior vena cava is based on the fact that the hydrostatic pressure necessary to dilate the auricle with its very thin wall is not adequate to produce sufficient intracaval pressure to insure its retrograde distribution through lumen decreasing tubes to the capillary bed. This is in

accord with the principles of the hydraulic gradient (20).

From this we may infer that two important purposes may be served by occluding the vena cava at its entrance into the auricle. First, the auricle will be relieved from its distending pressure, and second, each vena cava will be individually isolated and occluded to the end that its retrograde filling will more rapidly and completely retard the emptying of the arterial capillaries.

With the first experiments which were performed, it was obvious that, with present methods of constriction of the pulmonary artery and aorta, there are several serious handicaps, the results of which might well be overcome if it were possible to interrupt all circulation on the venous side in contrast to the blockage on the arterial side of the heart.

The most obvious insult at first apparent is the enormous distention of the right heart which occurs with the application of a temporary ligature to the pulmonary artery and aorta. The heart dilates rapidly when such a constrictor is applied. The rate increases at first; the force is greater; the heart seems to churn the blood and with each beat grows larger. The distention of the right ventricle and auricle then occurs, thus filling them to an enormous size with dilatation of the venæ cavæ. Extra systoles appear and the rhythm soon becomes very irregular. Gradually the force of the contractions weaken, the rate slows, and the heart stops in complete diastole.

The amount of force necessary to hold a constrictor on these vessels against the heart action of a normal dog is surprisingly great. Just as in any other muscle which has become fatigued by its contractions, this point is reached much sooner in the muscle which is stretched. The recovery phase of a fatigued muscle is greatly prolonged by continued efforts at contraction. Physiologically, as was first shown by Bowditch, we know that the contractions of the heart muscle are always maximal. They will be more quickly exhausted against an obstruction than a skeletal muscle which does not always respond with its greatest effort. We would expect to find that the heart muscle which had been exhausted by such a method would respond poorly to stimuli. It seemed logical, therefore, to propound that obstruction to the venous return to the right heart would prevent the extreme exhaustion of the myocardium as described and allow the normal rhythm of the heart to continue. This should favor the prompt recovery of the normal heart.

The physiological effect of distention of the right auricle was first shown in part by the work of Gaskell in dividing the auricle of the turtle heart into strips and then demonstrating that stretching of the auricular musculature greatly slows and finally inhibits the conduction of an impulse from the sino-auricular to the atrioventricular node.

More recently Lundy and Woodruff have clearly demonstrated the effect of right auricular distention by inserting a balloon into the auricle and distending the balloon under controlled pressure. They demonstrated that with intra-auricular pressures of 15 to 60 millimeters of mercury, there was a gradual lengthening of the P. R. interval from 0.08 second to 0.2 second. A period of 2:1 heart block, establishment of an independent ventricular rhythm and finally complete suppression of the P wave occurs.

After release of the intra-auricular pressure there was a gradual return to the control type of sinus rhythm with normal P. R. intervals.

Any increased auricular pressure directly affects the transmission of impulses from the sino-auricular node to the auricular-ventricular node through the media of the auricular musculature. It would seem logical, therefore, that the degree of cardiac irregularity will be directly proportional to the extent and duration of cardiac distention. Any prolongation of this interferring mechanism will undoubtedly prolong the time of recovery.

To obstruct the venæ cavæ would not only prevent an increase in the distention which had already occurred from interference with egress through the pulmonary artery partially occluded by an embolus but following incision of the pulmonary artery there would occur a relief of right heart distention. This relief should facilitate the return of right heart musculature to a more normal potential.

A B C

Fig. 2. Schematic drawings of stages in the development of the human heart. A (After Mall), 2.0 millimeters. B and C (After His), 2.2 millimeters and 3 millimeters. (From Arey's *Developmental Anatomy*. Courtesy, W. B. Saunders Company.)

This hypothesis is supported by the distention experiments of Lundy and Woodruff.

After holding a constrictor around the two great arteries and realizing the pull necessary to stop the blood flow from an actively beating heart, it is easy to understand why the intima of the pulmonary artery and even the pulmonary artery itself has ruptured in certain cases in doing a pulmonary embolectomy. When the heart action has been strong this has happened in cases operated upon by Nyström and also by Sauerbruch, the result being a subsequent thrombosis of the pulmonary artery and loss of the patient, or an uncontrollable hemorrhage from rupture of the artery. Such fatal results from otherwise successful cases would be unnecessary if it were possible to apply constriction on the venous side of the heart where the lightest force might control the flow of blood without danger to the vessels or the heart from pressure of the constriction.

Therefore it was concluded that if a method could be demonstrated whereby the interruption of the circulation might be made on the venous side of the heart we might expect the following results:

1. The time of interruption of the circulation might be safely prolonged.
2. Irreparable damages of the heart and brain should occur less frequently.
3. Distention of the right heart which occurs from obstruction of the pulmonary artery would be relieved.
4. Heart block or the arrhythmias occurring from right auricular distention could be avoided.
5. Following incision of the pulmonary artery in doing an embolectomy, the right heart distention could be relieved and the normal cardiac rhythm be continued or regained.
6. Recovery time for resumption of heart function following constriction should be reduced.
7. The danger of rupture of the pulmonary artery would be avoided.

EMBRYOLOGY OF THE HEART

The embryological development of the heart is of particular interest in this problem. To know the route of development by which the sino-auricular node comes to its final location will perhaps more clearly visualize for us the reason for interference in the conduction mechanism which occurs from distention of the right auricle. The evagination of the aortic bulb to form the ventral aorta and the pulmonary artery from a common origin also shows us why the transverse sinus comes to be so conveniently located in relation to our particular problem. Therefore we will consider the embryology of the heart in some detail.

As given to us by Arey's *Developmental Anatomy* the heart of the lower fishes and Amphibians develops directly within the ventral mesentery of the foregut, and the mesenchymal cells differentiate directly into endomyo and epicardial layers. Early heart development in lower forms such as fishes, reptiles, and birds is more complex.

The mesodermal elements very early appear between entoderm and splanchnic mesoderm and the elements arrange themselves into two

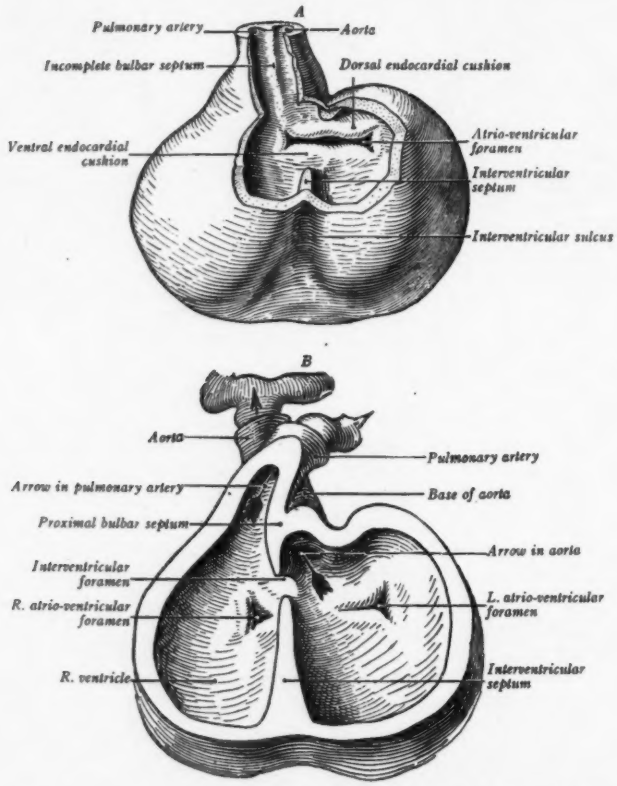

Fig. 3. Schematic ventral views of the opened human heart to show the division of the bulbus and the partitioning of the common ventricle (Kollman). A, At 5 millimeters; B, at 7.5 millimeters. (From Arey's *Developmental Anatomy*. Courtesy, W. B. Saunders Company.)

thin walled endothelial tubes, which soon fuse and develop a dorsal and ventral mesenteric attachment. The ventral mesenterial attachment presently disappears and the heart is left suspended in a pericardial chamber by a temporary dorsal mesocardium which is lost before the heart develops greatly. The endothelial tube forms the endocardium; the splanchnic mesoderm later gives rise to the epicardium and myocardium. This type of heart shows three regions: (1) the atrium, which receives blood from the primitive veins; (2) the ventricle; (3) the bulb, from which is given off the ventral aorta and the pulmonary artery.

Mammalian and human hearts also develop as two tubular cavities which unite. As the cardiac tube grows more rapidly than the pericardial cavity in which it lies, it bends to the right, throwing the bulb and the thick walled ventricular limb into a U-shaped loop. A fourth region is now distinguishable, the sinus venosus. The two horns of the sinus venosus soon become very unequal in size, the right side becoming very large and the left very small, due to the shift in flow of venous blood through the liver. This occurs by the time the embryo is 6 weeks old.

The sinus venosus opens into the right dorsal wall of the atrium and then follows a growth of the atrium laterally to the right and the left in two sacculations which are later to become right and left atria.

The bulb and ventricular limbs fuse to one cavity and in the 5 millimeter or 5 weeks old embryo, the heart is composed of three undivided chambers: (1) sinus venosus, (2) bilaterally dilated atria, (3) primitive undivided ventricle. The three-chambered heart is developed in adult fishes but in birds and mammals a four-chambered heart is developed in which venous blood circulates on the right side and arterial blood on the left side.

The heart of an embryo of 2 months has attained its general structural characteristics.

There remain four important steps to the formation of the human heart: (1) complete division of the atrium and ventricle into right and left sided chambers; (2) the incorporation of the sinus venosus into the walls of the right atrium; (3) the longitudinal division of the bulb and its distal continuation, the truncus arteriosus into the pulmonary artery and aorta; (4) the development of the semilunar and atrioventricular valves.

The second and third changes, as listed, are of interest to us in our particular problem. In embryos of 6 to 8 weeks, the atria increase rapidly in size and the lagging right horn of the sinus venosus is taken up into the wall of the right atrium. By this absorption, the superior vena cava of necessity drains directly into the cephalic wall of the atrium and the inferior vena cava into its caudal wall.

Pulsations in the human heart begin to appear at about the end of the third week, according to Simpkins, the sequence of contraction proceeding from the sinus to the primitive atrium. The first pulsation of the human heart has never been observed, but it probably begins very much as it does in the chicken, in which the initial pulsations occur as little tremors or jerks irregular at first and then settling down to a rhythmical pulsation.

The site of the pace-maker in the embryo heart has remained in the region of the sinus venosus from the time of the first primitive beats and does so as long as the normal heart mechanism persists. The relative position has shifted only in relation to the developing portions of the heart. The sinus or sino-auricular node finally comes to be situated at the junction of the superior vena cava and the free border of the right auricular appendage and extends down along the sulcus terminalis for a distance of 2 centimeters. The node is 2 millimeters in width and has a special arterial supply, which breaks up into a fine capillary network of nerve cells and fibers forming the excitor relay from the vagus nerve. Excitor fibers of the sympathetic are also present.

The mode of spread of the cardiac impulse has been worked out by studies of electrical variation in the beating heart. As the impulse is set up in the sino-auricular node it spreads out uniformly over the walls of both auricles, as when a stone is thrown into a pond and the waves spread concentrically.

The development of the pulmonary artery and aorta continues from the aortic bulb (which includes the truncus arteriosus) in the 5 millimeter embryo from bilateral thickenings which appear in the lumen of the aortic bulb. These thickenings meet and fuse to form two vessels, a dorsally placed aorta and a ventrally placed pulmonary artery. At the same time the interventricular septum is formed so that with a slight clockwise rotation which takes place the pulmonary artery unites with the right ventricle and the aorta unites with the left ventricle.

Since the pulmonary artery and aorta have a common embryological origin from the aortic bulb we find them in a common complete sheath of the visceral layer of the serous pericardium for a short distance, where the visceral layer was attached and made a compensatory growth with the development of the aortic bulb.

When the pericardium is opened from the front it is then found possible to pass the fingers or instruments behind these vessels in front of the atria through what is called the great transverse sinus. This is formed by reason of the reflection of parietal pericardium to envelop the heart.

MATERIALS AND METHODS

Three groups of animals were studied in this work. In the first group were 4 dogs each of which was operated upon and a constrictor applied to its pulmonary artery and aorta after the method of Trendelenburg. This was

done to study the reactions of the animals and to observe the effects on the heart, especially its change, contour, rate and rhythm, with the constriction by the method of Trendelenburg now used in the operation of pulmonary embolectomy.

In the second group were 3 animals in which a constrictor was applied to the superior vena cava with the view of preserving the respiratory center by maintaining a higher pressure in the capillary bed. This method was soon given up because it seemed to be impractical for either experimental purposes or human application.

In the third and largest group in this series the experimental work was done on 15 normal dogs. In this group the constrictor was applied in such a manner as to obstruct all return venous flow to the heart but no constriction was made of the pulmonary artery or the aorta.

The operative technique used in the preparation of these animals was done under nembutal anesthesia which was given in amounts of 1 cubic centimeter per 7 pounds of weight. Aseptic technique was carried out in only 5 cases, the remainder being done as acute experiments. A three way cannula was placed in the left external jugular vein on each animal and connected to a mercury manometer to study the back pressure developed in the venous system and its relation to the effect on the brain when constriction was applied to the great vessels of the heart.

A three way cannula was inserted in the left femoral artery and hooked to a mercury manometer to study arterial pressures when the great vessels were blocked, and during the recovery periods of the heart after constriction. Sodium citrate solution, 10 per cent, was used to prevent coagulation during the experiments. The lesser dilutions would not prevent clotting.

A left parasternal incision was made from the second to the sixth ribs about one-half inch from the midline. The costochondral cartilages were divided close to the sternum and the ribs retracted. The internal mammary vessels were ligated, after which the left pleura was incised. All dogs were given artificial respiration of air through a cannula in the larynx as soon as the pleura was incised. The pericardium was exposed and two stay sutures were placed on the ventral surface 1 centimeter apart and the pericardium was opened widely between them. These sutures were held to maintain a pericardial bed for the heart.

For the dogs in group I, a constrictor of narrow binding tape was used which was easily passed through the great transverse sinus by means of a special sound made in the shape of a large curved needle of No. 6 copper wire. Tightening of this tape made constriction of the pulmonary artery and the aorta.

The same type of constriction was used in the second group as in the first, plus a second tape which was placed about the superior vena cava. This caused the opening of the right pleura as it was opened in passing the tape about the superior vena cava with an aneurism needle.

For the third group of cases a special, large, double curved sound was made of copper wire which allowed passage of a constrictor through the transverse sinus from right to left, was then passed inferiorly to clear the pulmonary veins and then curved back to be brought out to the right side of the heart over the inferior vena cava. Although with practice this maneuver was easily performed, it seemed subsequently that the tissues could be more gently handled to pass a small rubber tube through the transverse sinus by means of the Trendelenburg sound and then slip the tube over the apex of the heart and gently draw it into place about the junction of the auricle and the inferior vena cava below and through the auricular appendage above. When the constrictor was tightened, it not only occluded the return circulation from the venæ cavæ but also that from the pulmonary veins. It was also easy to pass a new No. 16 male catheter through the transverse sinus with only the right index finger being used as a guide for the catheter. The finger was passed through the sinus from left to right, and the catheter was allowed to follow through from right to left. The catheter was found to be the proper size constrictor for use in our work on dogs.

RESULTS OF EXPERIMENTS AND OBSERVATIONS

In the following pages are appended the protocols of the animals which were used for the operative and experimental work on this problem.

Dog. 1. A white spotted short haired male setter, weighing 38 pounds was used for this experiment. A normal tracing after inserting cannulas showed a rate of 102, with a blood pressure of 50 millimeters' mercury. After the chest was opened under artificial respiration the blood pressure was 56 millimeters and the pulse 162.

During the placing of a constrictor, there was considerable manipulation necessary, with a rise in blood pressure to 77 millimeters with a gradual fall to 61 millimeters over a period of 1 minute and 10 seconds.

When the constrictor was applied, there was an immediate fall in blood pressure to 11 millimeters of mercury and a rise of the venous pressure of 6 millimeters. There followed a gradual drop in arterial pressure during a period of 30 seconds' constriction to 5.5 millimeters and on release of pressure an additional drop to 3.5 millimeters for about 2 seconds. There was no cessation of regular beating but the rapidity of distention of the heart and the force of contraction was surprising. There was especially great distention of the right heart. The corneal reflexes remained normal and the dog had no convulsions. The coronary vessels showed an enormous distention with increased tortuosity.

Following release of constriction, the heart continued with its regular rhythm and with the first full contraction the blood pressure rose from 3.5 to 18 millimeters. With a regular rate of 132, there was a gradual rise in pressure to 70 millimeters. It should be pointed out that this return to normal is much slower than the return of the normal or even of this dog (No. 1A), when constriction was applied to the afferent vessels.

The chest wall was closed in layers, it being impossible to close the pleura. As the last sutures were put in place, the lungs were distended by positive pressure to fill the chest and expel all the air possible. Following closure artificial respiration was stopped and, after 45 seconds, the dog began breathing normally, save for some increased use of the accessory muscles, then it became regular, short and labored. Pulse was 154 but of fair quality. Sheets and heat were applied for a short time, after which the animal was covered and placed in a cage. In the cage the dog appeared to be in mild shock with a pulse of 164 and thready. He reacted from the anesthetic.

On August 5, 1933, he was up in the cage, appeared fairly comfortable, and was friendly. He ate ⅓ can of dog-meat with good appetite, and drank some water. His respiratory movements and panting were apparently normal. There was a slight semipurulent discharge from the left eye.

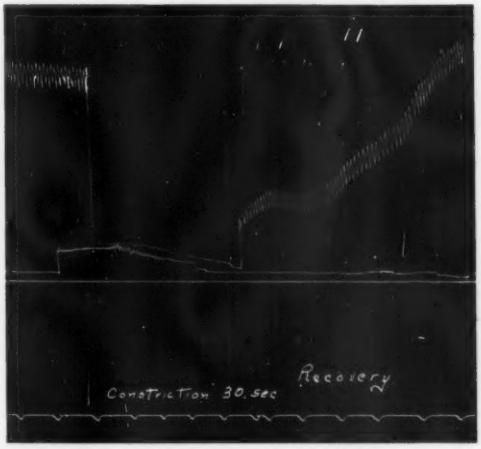

Fig. 4. Dog. 1. Kymographic tracing showing the slow recovery time of the normal heart when constricted by the Trendelenburg method for only 30 seconds. Compare with Figure 7.

The dog remained well except for a hematoma which formed at the cephalic end of the wound which was drained August 11, 1933, and which continued to drain for about 2 weeks. He lost 6¾ pounds in weight, weighing 31¼ pounds on August 14, 1933. This dog was kept and a second operation performed December 13, 1933.

Dog 5. A brown long haired female dog weighing 34¾ pounds was used in this experiment. Constriction was applied to the pulmonary artery and aorta, and an ineffectual attempt was made to control the incoming flow of blood to the auricle by digital compression. With application of the constrictor, the heart action became forceful, increased in rate, and with each full powerful beat dilated noticeably. The heart continued with a regular rhythm for 1 minute and 20 seconds, then a few extra systoles appeared. From this time on the force of contraction became weaker with each beat and seemed inversely proportionate to the amount of dilatation. Constriction was held effectively as checked by the manometer for 3 minutes, during the latter part of which the heart was greatly dilated and contractions feeble. Seventy seconds after release of the constrictor, ventricular contractions ceased and the heart distended to its greatest capacity and the musculature remained perfectly flaccid. Five minims of adrenalin (hydrochloride) was given in the right ventricle with no evident effect. One cubic centimeter more of adrenalin was given into the base of the aorta and almost immediately ventricular fibrillation was seen to start in the right ventricle and spread over the whole ventricular musculature. Following a post-mortem examination of this animal, I felt that by digital compression the right auricle could be compressed so as to prevent return flow of blood to the

heart from the vena cava. It seemed obvious that the extreme dilatation with the strain of stretching of the musculature made resuscitation after constriction difficult, if not impossible.

Dog 6. A large brown mongrel female weighing 32 pounds was used for this experiment which was conducted with the usual set-up and with a constrictor around the pulmonary artery and aorta through the transverse sinus. Digital compression of the right auricle was made with the auricle between the extended index and middle fingers by adduction of the fingers at the same time as the constriction of arteries. The constriction of the pulmonary artery and aorta was held for 2 minutes and 40 seconds total time. I was determined to stop constriction short of a lethal time for the dog. With constriction on and compression of the auricle, there seemed to be no less dilatation than without compression. The pupils dilated at 30 seconds and at 1:45 seconds were widely dilated. Corneal reflexes failed at 1:50 seconds. At 1:58 the animal had one mild convulsion followed by a second seizure of about the same type.

The heart was beating with good force in spite of the dilatation after 2 minutes' constriction, although the rhythm was somewhat irregular. The right heart was more dilated than the left, the line of the interventricular septum being plainly seen. It was felt that constriction could be safely carried longer. At 2 minutes and 30 seconds, contractions became rapidly weaker and quite irregular with a 2:1 heart block. At 2 minutes and 40 seconds, the ventricles ceased to contract and as soon as the constrictor was released fibrillation was seen over the right ventricle which spread rapidly to both ventricles.

Dog 7. A large mongrel male dog weighing 45 pounds was used for this experiment. The usual set-up of apparatus was used and a constrictor was placed around the pulmonary artery and aorta through the transverse sinus. An attempt was made to put digital compression on the right auricle to prevent the dilatation of the right heart but without evident effect. A normal blood pressure tracing showed the pressure to be 76 millimeters of mercury and after opening the chest and placing the constrictors, the pressure was 30 millimeters. This was a greater fall in pressure than was usually seen. The venous pressure failed to register.

Constriction was held for 2 minutes with the usual dilatation of the heart which usually occurs following the first minute of active beating when a constrictor is applied to the great arteries. The arterial pressure made a gradual fall to a 4 millimeter level in 25 seconds, after which followed a period of 30 seconds in which a high pulse pressure was present. The rate during this period was 74; rhythm regular. The heart apparently made a normal recovery.

Much to my surprise, while the chest was being closed, the heart was seen to be in a state of ventricular fibrillation. This time was estimated to be about 6 minutes after the time of the 2 minute obstruction to circulation.

Dog 2. A medium sized black and brown mongrel dog, weighing 28 pounds, was used for this experiment. A normal kymograph tracing showed the blood pressure to be 52 millimeters mercury with a rate of 84. After the chest was opened and the constrictor placed about the pulmonary artery and aorta, a second tape was placed about the superior vena cava. This was done by opening the right pleura and passing a threaded dull aneurism needle through the pleura into the posterior mediastinum and bringing one end of the tape out to the left of the superior vena cava. Constriction was then applied to the pulmonary artery, aorta, and superior vena cava simultaneously to stop all circulation for 3 minutes.

The heart was very active for 1 minute, beating wildly with full powerful strokes and with what appeared to be an occasional extra systole. The pupils were dilated well at 45 seconds and at 1 minute, dilatation was complete. At 2 minutes and 10 seconds, the animal staged a mild convulsion followed in succession 10 and 15 seconds apart by four hard seizures. The force necessary to hold the arterial circulation for the first 1 minute was very great, after which the force of the contractions lessened and the heart began to dilate, at first slowly, and then as the rate began to slow, dilatation became extreme. At 2 minutes and 48 seconds, the beating ceased. The venous pressure made a gradual rise for 1½ minutes during constriction to a level of 9 millimeters where it remained until release, except for the rises occasioned by the convulsive seizures. After 3 minutes of constriction, pressure was slowly released and feeble cardiac contractions began immediately—at first one beat every 3 to 4 seconds, then, taking on an irregular rhythm with acceleration of the rate and an increasing force, the heart came to a normal size and rate in about 30 seconds. The rate and force with a regular rhythm then continued to rise to exceed normal, then gradually slowed to a rate of 104 after a 1 minute, 20 second period.

The pericardium was closed loosely and the chest in layers with no attempt made to close the pleura. Artificial respiration was stopped after the chest was closed and the dog began shallow regular respirations 30 seconds later.

The following day the dog looked well, ate some finely ground meat, and drank a little milk. He was up in the cage and friendly. The third day after operation he was listless, hot, and did not drink. The following morning he was dead.

Autopsy showed a collapse of both lungs with no air containing tissue except at the right upper lobe. The pericardium was not distended because it was closed loosely; the serous surface was smooth but dull.

Dog 3. A long haired black and white mongrel female dog weighing 32¾ pounds was used in this experiment. The set-up of apparatus and operation was made as has been described and constrictors were placed around the pulmonary artery and aorta and the superior vena cava. A normal pressure tracing showed the arterial pressure was 65 millimeters

before the chest was opened. After the constrictors were in place the pressure was 44 millimeters.

Constriction was applied for 3 minutes. Heart action was strong and regular with some dilatation for 1½ minutes, and then followed marked dilatation, arrhythmia, gradual slowing and loss of force of contraction. The ventricles stopped beating after 2:55 seconds of constriction from exhaustion in beating against the obstruction, the heart being apparently stretched to its greatest capacity, while the auricles continued with a regular rhythm. During constriction, the arterial and venous pressures equalized and after 1½ minutes showed equal levels, according to measurements, varying from 6 to 9 millimeters of pressure.

On release of constriction the ventricles made a few feeble beats for 15 seconds and then stopped. Dilatation was extreme. Cardiac massage was tried without response. Adrenalin 0.5 cubic centimeter was injected into the root of the aorta without effect. One cubic centimeter of adrenalin was then injected into the right ventricle which was followed by a wildly violent response. The manometer was pushed off the drum and blood was forced all the way through the manometer tubing and part of the mercury pushed out of the manometer. The tracing shows a pressure of 105 millimeters but was much higher and the manometer had to be clamped off.

The total time that circulation was suspended, except for the result of cardiac massage, was 5 minutes. After the chest was closed artificial respiration was stopped and normal respirations began at once. Pulse was 142 and of good quality.

The following morning the dog was dead. There was a complete atelectasis of the left lung and nearly complete on the right except for a few air containing areas in the middle and lower lobes. About 10 cubic centimeters of bloody serous material was present in each pleural cavity. The right heart was widely dilated. No gross changes were present in the brain.

Dog 4. A large male police dog weighing 46 pounds was used for this experiment. The set-up of apparatus and operation was as has been described with two constrictors placed around the pulmonary artery and aorta and the superior vena cava. The normal blood pressure was 54 millimeters and, after the constrictors were in place, was 42 millimeters. Constriction was simultaneously applied with the two constrictors for 3 minutes. In this instance heart action was at no time entirely discontinued, but after 2 minutes and 10 seconds contractions were feeble although carried on at a regular rhythm. The effect of sudden release of the constrictor was seen here as the heart seemed to lose its rhythm and was overwhelmed for a few beats. The rate and acceleration were slow, but with strong powerful beats the heart action climbed to a rapid rate and a forceful normal rhythm. Pressure returned 40 to 50 millimeters mercury. After the chest was closed respirations began a short time after the tracheal cannula was removed. He was returned to the cage apparently in good condition. The venous pressure showed a rise to 13 millimeters during the time of constriction.

After operation he looked fairly well for 2 days but apparently had a poor appetite. The morning of the fourth postoperative day the dog was found dead. He had a marked fibrinopurulent pleurisy with a complete left sided atelectasis. The right lung showed atelectasis with partial consolidation of the right lower lobe. The pericardium was distended with a thin purulent fluid with a thick fibrinopurulent exudate on the epicardium.

Dog 8. This was a young police dog weighing 30 pounds, and the usual set-up of apparatus was made. It had been found by previous experiments on dead dogs that a tube or tape might be passed through the great transverse sinus and then by swinging the sound carrying the tape up to the right side of the apex, the constricting tape would surround the right auricle and the pulmonary veins and thus shut off all return circulation to the heart when tightened and used as a constrictor. In order to obstruct all the vessels, it was necessary to tighten the tape against two or three opposing fingers of the opposite hand.

A sound with a double curve was made of heavy copper wire to carry this constrictor tape. To facilitate the passage of this sound for the first time a suture was placed in the apex of the heart and traction made on the heart to stabilize it and pull it somewhat to the left. The sound was easily passed and the constrictor put in place. A tape was also put around the pulmonary artery and aorta in the usual manner. Constriction was applied to both the pulmonary artery and aorta and the right auricle for 2 minutes. The heart maintained its normal contour and its normal rhythmical contractions during the whole time the tapes were tightened. The dog had no convulsions.

On release of constriction the whole heart, especially the right, filled up rapidly like a balloon and the contractions of the ventricles became feeble and irregular. The heart seemed to be overwhelmed with no ability to recover its normal forceful rhythm. After 4 minutes and 22 seconds the heart went into ventricular fibrillation and the dog expired.

Dog 9. This was a large white mongrel dog weighing 45 pounds. The usual set-up of apparatus was made and two constrictors were placed as in the previous experiment.

Circulation was obstructed by tightening both constrictors for 2 minutes, after which time the tape about the pulmonary artery and aorta was loosened. It was surprising when no change occurred in the arterial or venous pressures and the contour of the heart, its rate and rhythm remained unchanged, until it was realized that the tape around the auricle prevented all blood flow to the heart. There was no dilatation of the heart. The tape was held on the auricle for an additional 65 seconds. The venous pressure rose to 7 to 8 millimeters and the arterial pressure fell to 7 millimeters during the circulatory obstruction.

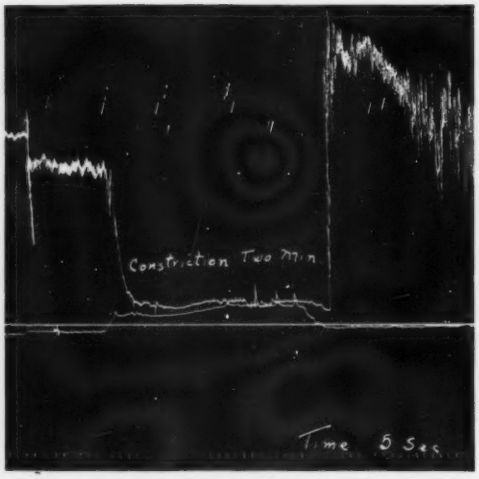

Fig. 5. Dog. 10. Kymographic tracing showing rapid recovery of the normal heart when the afferent vessels are constricted. Compare with Figure 4.

When the auricular obstruction to circulation was released, the right heart dilated widely but the regular rhythm supervened and the blood pressure rose in 5 seconds to 82 millimeters. This pressure was maintained for 5 minutes, and then slowly fell to 61 millimeters.

The chest was closed. When artificial respiration was stopped the dog breathed at once. The next morning he was found dead, having succumbed to a pneumothorax. The right pleura was intact at postmortem, but a massive atelectasis of both lungs was present.

Dog 10. This was a white long haired male mongrel dog which weighed 20 pounds. The usual set-up of apparatus was made and constrictor was placed about the right auricle by use of the special sound.

The circulation was obstructed for 2 minutes. The heart remained normal in size and maintained its normal rhythm for the full time of obstruction. When the tape was loosened the right heart dilated for about 5 seconds but the normal rhythm continued and the heart took up its work immediately rising to 78 millimeters' pressure in 6 beats.

The normal blood pressure was 65 millimeters. After the chest was opened and the tape in place, the pressure was 55 millimeters. During circulatory obstruction, the arterial pressure was 7 to 9 millimeters while the venous pressure was of equal height. At 1 minute and 30 seconds the dog had a hard convulsion followed by two somewhat milder seizures 15 seconds apart. During the first convulsion the venous pressure rose to 17 millimeters mercury. It was interesting to note the oscillations of the arterial manometer during constriction of the auricle.

The chest was closed and respirations started 1 minute and 25 seconds after artificial respiration was stopped. When returned to the cage, the dog was cold, pulse was 172 and weak, and respirations rapid and shallow. He was wrapped up and heat lamps applied for a short time. The next day he was not active, would not eat or drink. Respirations were rapid and shallow. The following morning he was dead. A bilateral atelectasis was noted—the right lung having a little air containing area in the right upper lobe, the atelectasis being complete on the left side.

Dog 11. A large brown long haired male dog weighing 50 pounds was used for this experiment. The usual manometers were hooked up, the chest opened, and a constrictor passed about the right auricle by means of the special sound. The normal blood pressure level was 65 millimeters of mercury. After the chest was open and the constrictor in place the pressure was 38 millimeters.

Circulation was obstructed for 3 minutes, by tightening the tape around the right auricle. During this time the heart maintained its contour without dilatation and regular forceful contractions of the heart continued. After 1 minute and 33 seconds' obstruction the arterial pressure fell from its usual level of 4 millimeters to a −1.5 millimeters where it remained until release. The right heart dilated for 30 seconds following release of the right auricle during which time the arterial pressure was 7 to 9 millimeters, but the rate was 100 and the rhythm regular. The force of contraction was too weak to raise the pressure. After this latent period of recovery, the pressure started to rise and with a high pulse pressure the pressure gradually rose to 78 millimeters in 90 seconds, the venous pressure showing a gradual fall.

After a recovery period of 5 minutes, constriction was reapplied for 1 minute during which time the ventricular rhythm was very irregular. The heart did not dilate. The force of contractions of the heart became weak, and the rate rapid so that it was thought advisable to release the pressure. The pressure rose to 59 millimeters almost at once but fell in 15 seconds to 35 millimeters during which time the auricles were in a state of flutter and the ventricles were beating wildly with a rate of 226. The apex of the heart seemed to contract normally but the ventricles toward the base seemed to be in a fibrillatory state, which lasted for 1½ minutes.

Three minute recovery periods were allowed following constriction. Three short periods of obstruction were maintained for 1½ minutes, 1½ minutes, and 2 minutes. The recovery periods were rapid and the right heart showed only a temporary dilatation for a few beats on release of the constrictor. Then followed 5 successive constriction periods of 2½ minutes each with recovery periods of 5 minutes between obstructions of the circulation. When the constriction was released the regular rhythmic contractions were temporarily slowed by a dilatation of the right heart. The heart made a few full strong contractions, the pressure rose rapidly, and the rate accelerated, timed at 152 to 160 on several occasions after reaching a stable rate, rhythm, and pressure.

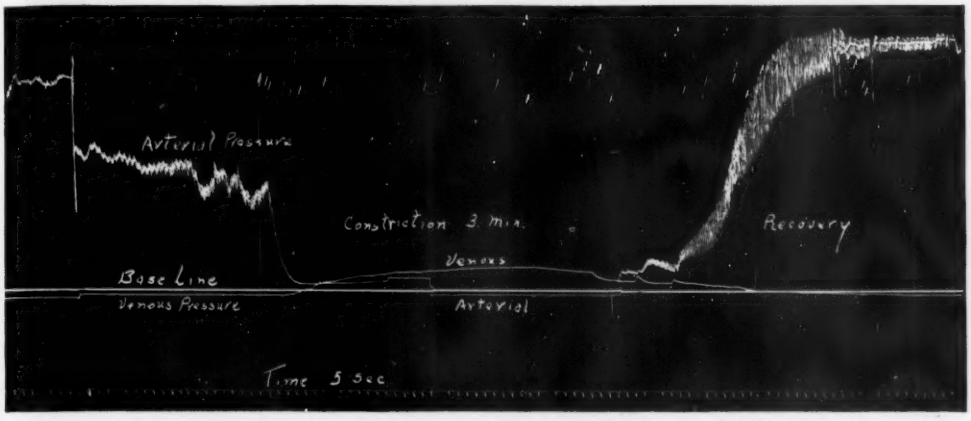

Fig. 6. Dog 11. Showing a latent period of recovery of the heart following constriction at the auricle.

When obstruction periods totaling 14 minutes were passed, the artificial respirator was disconnected and the dog began to make slow respiratory efforts. This also happened as the experiment terminated.

A total obstruction of the circulation for 21½ minutes was made on this dog at interrupted intervals, the longest of which was 3 minutes and the shortest of which was 1 minute.

Dog 12. A healthy appearing male police dog weighing 32 pounds was used for this experiment. A complete protocol is given on this dog as being typical of the observations and the work done on this particular group. On the others less detail need be given. The usual set-up of apparatus and exposure was made and a constrictor passed around the right auricle. Some difficulty was experienced in passing the sound because the transverse sinus was smaller than usual, admitting the index finger very snugly. The blood pressure which was normally 61 millimeters of mercury, after the constrictor was in place, was 44 millimeters.

The circulation was obstructed for 2 minutes by tightening the tape about the auricle. The heart continued to beat normally without distention for 1 minute and 40 seconds, and then the right heart began to dilate somewhat and the contractions became slower and weaker.

Physiological Ringer's solution (Locke's formula) (17) had been made up and was freely applied to the heart during the period of obstruction to prevent drying and with a hope of giving an added stimulus to the myocardium. The solution was made up as follows:

	Per cent
Sodium chloride	0.9
Calcium chloride	0.24
Potassium chloride	0.42
Sodium bicarbonate	0.02

As soon as the pressure was released without any evidence of temporary fall in pressure the heart took up a forceful beat. In two contractions of the heart the pressure rose from 6 millimeters to 53 millimeters and then continued to rise with a high pulse pressure. In 25 seconds the pressure rose so high that the manometer indicator was forced off the kymograph and with high oscillations went as high as 42 millimeters above the tracing. This made a pressure of 149 millimeters which was higher than any previously seen normally or during the recovery stage. The arterial pressure fell to a pressure level equal to that of the venous pressure which varied from 4 to 7.5 millimeters during constriction, there being a slow rise of 3.5 millimeters after an initial low 35 seconds after the application of the constrictor.

The constrictor was again applied 4½ minutes after release of the first period. This time there occurred an almost identical course with the first except the ventricular contractions which were maintained with a regular forceful rhythm slowed only slightly at 2 minutes and 30 seconds. Obstruction was released at 2 minutes and 30 seconds and in 5 seconds the pressure started to rise from full equal beats. The pressure rose to 116 millimeters and gradually lowered in 3½ minutes to 54 millimeters. Ringer's solution was used less freely.

After a rest period of 5 minutes a third period of obstruction for 3 minutes was maintained. The ventricular contractions were maintained throughout, although they slowed at 2 minutes and 30 seconds and the force seemed somewhat weaker to the hand holding the constrictor from then until release. On release there followed a prompt recovery but this time with a rise in blood pressure to only a normal level of 60 millimeters and a gradual fall to 46 millimeters in 3½ minutes.

A fourth period of constriction was maintained for 4 minutes. At 3½ minutes, the ventricles were beat-

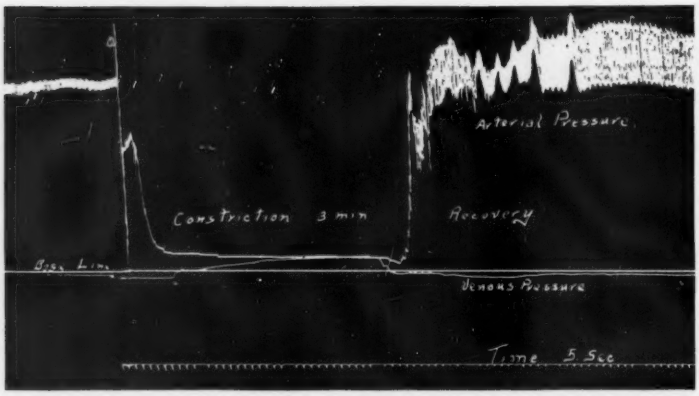

Fig. 7. Dog 1A. Kymographic tracing showing arterial and venous pressures with constriction of afferent heart vessels for 3 minutes and the prompt recovery which follows. (Compare with Figure 4 made from same animal 4 months previously showing slow recovery when efferent vessels were constricted for only 30 seconds.)

ing slowly but contractions were strong, regular, and complete. The rate had obviously slowed. There was no distention. The dog staged one hard convulsion at 3 minutes and 40 seconds and his pupils were widely dilated with absent corneal reflexes. At 3 minutes and 43 seconds the right heart began to dilate and contractions to weaken although no evident change was made in the manner of constriction. When the tape was released the right auricle and ventricle were well distended but dilated widely on release. The heart began weak, very irregular beats without force and with no rise in blood pressure. After 35 seconds of these irregular feeble beats the ventricles began to fibrillate. Ringer's solution was freely applied during these periods.

This dog survived 7½ minutes of circulatory obstruction but not an additional 4 minute period.

Dog 13. A large, thin male police dog weighing 37 pounds was used. This dog had a rapid heart rate of 152 with irregular respirations before operation. The usual set-up and exposure was made with a constrictor tape applied to the right auricle. The artificial respirator was not working properly during manipulation in passing the sound and the animal developed a marked cyanosis and a 2:1 heart block lasting 45 or 50 seconds. Soon after the respirator was fixed and the heart was moistened with Ringer's solution, the rate picked up and the rhythm became regular.

Obstruction of the right auricle was held for 2 minutes. The heart remained regular without dilatation but the venous pressure rose 3 millimeters higher during compression than the arterial pressure. At 1 minute and 55 seconds the dog had one hard convulsion and following release the heart recovered promptly, the pressure rising to a high level of 98 millimeters. During a 5 minute rest period, the pressure fell to 38 millimeters.

Constriction was applied for 3 minutes. At 2 minutes and 10 seconds of constriction the heart beat became slow and irregular. The application of Ringer's solution was a decided stimulus to the heart, the rate increased to about normal and the rhythm again became regular. At 1 minute and 20 seconds the dog had a convulsion followed by another 23 seconds later.

Following release of the circulation, the heart took up a feeble regular rhythm with a blood pressure of 11 millimeters which lasted for 30 seconds without signs of recovery. Ringer's solution was applied freely and the heart immediately began more vigorous efforts, showing a gradual rise in blood pressure to 45 millimeters in 50 seconds. The continued application of Ringer's solution made the beat tumultuous although there was no greater rise in blood pressure. The rhythm returned to normal when no more solution was applied. The stimulation of this solution for the heart was demonstrated on this heart on three different occasions, during the course of this experiment.

A third period of constriction of 2 minutes and 45 seconds was followed by weak irregular contractions for 35 seconds, during which time the right heart was greatly dilated and after which the ventricles began to fibrillate while the auricles maintained a regular rhythmic beating.

Dog 1A. This dog was a brown and black white spotted male dog of the setter type which had previously been operated upon August 4, 1933, and the pulmonary artery and aorta clamped off for 30 seconds. The usual arterial and venous manometers were used and the chest was opened by a left parasternal incision from the second to sixth ribs. When the pleura was opened no adhesions were encountered. The right medial pleura was slightly thickened. The pericardium and epicardium were free and their sur-

faces shiny, with 2 to 3 cubic centimeters of serous fluid present in the pericardial sac. The former line of incision in the pericardium was covered by a thin layer of fat. The only adhesions present were in the great transverse sinus. Here the adhesions were firm and fibrous and some difficulty was encountered before these were broken and it was possible to pass a sound through the sinus. A tape constrictor was then placed about the right auricle and tightened for 3 minutes. The heart action remained strong and regular at the rate of 104 until 2 minutes and 45 seconds, when the rhythm became somewhat irregular. A prompt recovery followed with the heart beating regularly and its action strong. No convulsions appeared.

After 5 minutes, a second constriction period was held for 2 minutes. Heart action weakened at 1 minute and 28 seconds and became irregular at 1 minute and 40 seconds. The heart was not dilated and when released dilatation of the right heart occurred for a few beats but was overcome by a prompt recovery. His pulse following constriction was 152. Three interrupted sutures were placed in the pericardium and the chest wall was closed in layers. He was placed under lamps to warm him and then covered when put into the cage.

The following day he was out of the cage and around with the diener and seemed active and in no distress.

December 16, 1933, dog was active, respirations good and deep—rate 22 and pulse 112. There was a dependent edema and redness of the wound at the upper end, extending into the neck.

December 21, 1933, small abscess was draining at upper angle of wound. The dog was in good condition and active.

December 26, 1933, animal was not eating or drinking well, wound closing nicely.

December 30, 1933, dog was found dead in cage and was thrown out before autopsy could be done.

Dog 14. A small male wolf-dog weighing 28 pounds was used. The usual set-up of apparatus was used and a constrictor placed about the right auricle. Constriction was applied for 2½ minutes. At 2 minutes and 15 seconds contractions became irregular and rate slowed. At 2 minutes and 30 seconds, there was marked weakness of contractions and irregularity so that it seemed necessary to release the obstruction. Release was followed by feeble, irregular contractions which failed to raise the blood pressure. After 1 minute 0.5 cubic centimeter of adrenalin was injected into each of the right and left ventricles. A most marked response followed. The rate became very rapid and pressure rose to 168 millimeters of mercury. One convulsion followed 30 seconds after release of the constrictor.

A second constriction period of 2 minutes was maintained without event. The heart did not dilate. After 3 minutes' rest as the tape was tightened for a third period, the ventricles began beating wildly in a ventricular fluttering state. The tension was varied without change. After about 10 seconds the constriction was loosened and my fingers opposing the constrictor were changed, then the tape was immediately tightened, a normal rhythm followed.

In all eight periods, constriction was maintained in which all circulation was blocked for times of 2, 2½, 3, and 4 minutes. The time and order in which these periods came were:

Period	1	2	3	4	5	6	7	8	9	Totals	9
Time	2½	2	3	2	2	3	3	4	3 death		21½

After the fourth period of constriction, which was at the end of 9½ minutes of interrupted obstruction to the circulation, the artificial respirator was disconnected and 48 seconds later the dog began to make respiratory efforts. After the sixth period, contractions were weak and the heart dilated as soon as obstruction was released. After 30 seconds of feeble contractions 5 cubic centimeters of adrenalin was injected, one-half into each ventricle with immediate response but less active than with the first adrenalin given.

In all, the heart survived 21½ minutes of periodic obstruction. Following the ninth period of 3 minutes the heart showed a 2:1 heart block which was maintained for 3 minutes and followed by ventricle fibrillation.

Dog 15. A large male Russian wolfhound weighing 42 pounds was used for this work. The usual set-up was used and a constrictor placed around the right auricle and tightened for 3 minutes. The rhythm remained regular throughout. The heart remained normal in size and made a very prompt recovery on release of pressure. The normal blood pressure was 75 millimeters and when constrictors were in place was 55 millimeters. Following release the pressure rose to 114 millimeters, then gradually lowered to 51 millimeters. Ringer's solution was applied freely during this period of obstruction. The high pressure may well be due in part to this, for it was not used to more than moisten the heart in the next period. A 5 minute recovery period was allowed between periods.

A second period of constriction was applied for 4 minutes and a kymographic tracing was taken of this period. Except for a slight slowing of the rate after 3 minutes and 10 seconds nothing remarkable occurred. No convulsions appeared and a prompt recovery followed. Ringer's solution was applied to the heart to moisten exposed surfaces.

A third period of obstruction was applied for 4½ minutes. At 3 minutes there was a decided slowing and weakness of the heart beat. Ringer's solution was applied freely and the rate and force returned even stronger than the normal immediately following the application. The rate slowed slightly and the force of contraction became weak, remaining regular, but when the constrictor was released the recovery was prompt.

In the fourth and last period the circulation was obstructed for 5 minutes. Again a stimulus was seen from Ringer's solution when applied to a somewhat irregular and rapidly weakening heart after 2 min-

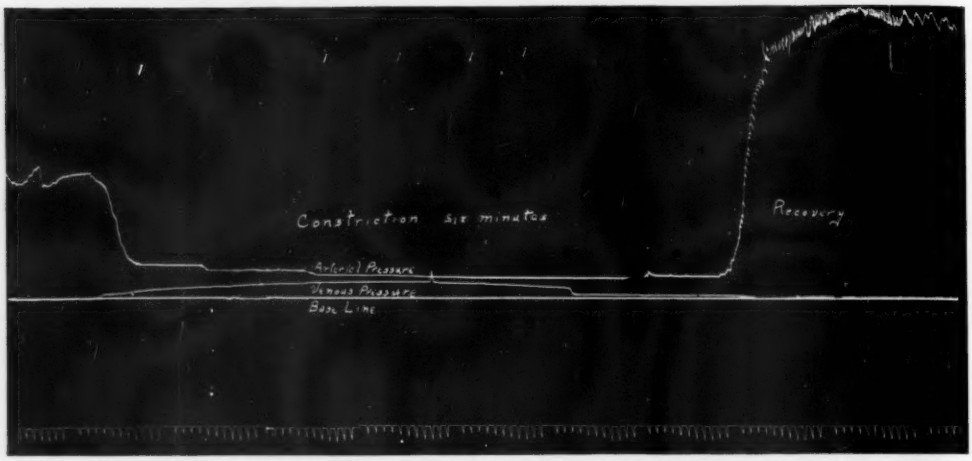

Fig. 8. Dog 17. Kymographic tracing showing the effects of constriction of the afferent heart vessels for 6 minutes, followed by a latent period of recovery of 50 seconds in which the heart beat regularly but weakly. Recovery followed an injection of 3 millimeters of adrenalin hydrochloride into each ventricle.

utes and 55 seconds. During the last 28 seconds the right heart dilated and the rate and rhythm became irregular again, while during the last 8 to 10 seconds the rate was very slow. Convulsions occurred at 2 minutes and 45 seconds and at 4 minutes. Recovery was prompt, and the experiment terminated. The circulation in this case was completely obstructed for periods of 3, 4, 4½, 5 minutes with 5 minute rest periods between, making a total time of 16½ minutes. When the artificial respirator was disconnected as the experiment was terminated, the dog made vigorous respiratory efforts for 2 minutes, 40 seconds, and at 3 minutes, 8 seconds two hard convulsions occurred.

Dog 16. A large police dog weighing 45 pounds was used for this experiment, and the cannulas were inserted in the vein and artery. Constriction was applied to the auricle for 2¼ minutes. At the end of 2 minutes the rate had slowed from 112 to 68 and the contractions were very weak. When the tape was released a prompt recovery followed. Ringer's solution was freely applied and the pressure rose to a height of 128 millimeters. Five minute rest periods were allowed.

Obstruction at the auricle was again maintained for 4½ minutes. Contractions were strong and regular for 2½ minutes, then gradually weakened and slowed but remained regular. Because of the weakness of contractions, constriction was released. Recovery was prompt. Ringer's solution only was used freely as stimulant. Respiratory efforts were noted throughout this period and one convulsion occurred as the constrictor was released.

Constriction was again applied for 4 minutes. The contractions soon weakened and then were stronger when Ringer's solution was applied. The rate again slowed and contraction weakened. At 3 minutes and 10 seconds a 2:1 heart block was noted. At 3 minutes and 35 seconds a 3:1 heart block was present and at 3 minutes and 53 seconds the heart stopped.

Release of constriction caused marked dilatation of the right heart and only irregular, slow contractions of the heart occurred. After 30 seconds adrenalin hydrochloride, 3 millimeters, was given into each ventricle. Ventricular fibrillation followed immediately after the adrenalin injection. The animal survived constriction of 6¾ minutes of obstruction to the circulation held in two periods, but failed to survive another 4 minute period.

Dog 17. A brown mongrel male dog weighing 36 pounds was used for this experiment. The usual setup was made and a constrictor placed about the right auricle. The constrictor was tightened and obstruction maintained for 6 minutes. The normal blood pressure was 52 millimeters and after the chest was opened and the constrictor in place, was 43 millimeters. With constriction, the arterial blood pressure fell to 7.5 millimeters and the venous pressure to 6 millimeters.

The heart rate was 118 during constriction and maintained a regular rhythm and normal size until the last half minute when the rate slowed and contractions weakened somewhat. Following release the heart beat feebly and showed no sign of recovery. After 50 seconds, adrenalin hydrochloride 3 millimeters was injected into each ventricle and an immediate recovery followed. The blood pressure rose to 97 millimeters and remained at 80 millimeters for at least 5 minutes. One convulsion occurred at 1 minute and a second hard seizure followed at 3 minutes and 25 seconds.

A second constriction period of 3 minutes was uneventful. For the third period of constriction, obstruction was maintained for 4 minutes. After 3 min-

utes and 10 seconds a 2:1 heart block occurred. When constriction was applied for the fourth period, the rhythm became so irregular it seemed wise to let the constrictor loose and reapply it. Obstruction was held 10 seconds then released, the fingers and constrictor changed in position, and then tightened and held for 4 minutes. At 3 minutes and 20 seconds, the rate was 46. The rate continued to slow but on release the heart recovered promptly. A 3¾ minute constriction period was held. The rate slowed gradually after 2 minutes and 55 seconds, being entirely stopped for the last 5 seconds. A prompt recovery followed.

Then followed a series of six 4 minute periods of obstruction to circulation except the ninth period was carried 4½ minutes. During the eighth period the rate slowed to 15 beats per minute.

After the last period the heart dilated and showed no sign of progress to recovery. After 2 minutes and 12 seconds 3 millimeters of adrenalin was given in each ventricle and the heart recovered a regular rhythm. The experiment was terminated voluntarily.

Period...	1	2	3	4	5	6	7	8	9	10	11	Total 11
Time....	6″	3″	4″	4′ 10″	3¾′	4	4	4	4½	4	4	Total 45′ 25″

Dog 18. A large police dog weighing 42 pounds was used for this experiment. The usual set-up was made and a constricting tape passed around the right auricle and held for 3½ minutes. The heart maintained a regular normal rate, did not dilate and on release took up its work without missing a beat. The right heart dilated moderately for a few beats immediately following release of constriction. A 5 minute rest period was allowed.

Constriction was again applied for 4 minutes. During the last 30 seconds, the right heart dilated widely without missing a beat. The right heart dilated moderately for a few beats immediately following release of constriction. A 5 minute rest period was allowed.

Constriction was again applied for 4 minutes. During the last 30 seconds the right heart dilated widely without change of pressure relations on the drum. On release of the constricting tape the heart dilated, made a few feeble contractions and started a ventricular fibrillation.

Dog 19. A large old police dog weighing 52 pounds was used for this experiment. The usual set-up was made and a constricting tape passed about the right auricle. The coronary vessels were thickened and tortuous in appearance.

Constriction was applied for 5 minutes. The heart action remained strong for 4½ minutes, although at 3 minutes and 12 seconds, the rate halved and a 2:1 block was noted. Contractions were strong and regular. At 4 minutes and 40 seconds the 2:1 block disappeared, the rate doubled, but the heart action was very weak. At 4 minutes and 45 seconds, the right heart dilated. At 4 minutes and 52 seconds, the ventricular action was stopped, the heart dilated and stopped in diastole. On release the heart distended widely and very weak irregular purposeless movements were seen. One-half cubic centimeter of ephedrin was injected into each ventricle and the heart immediately set up a ventricular fibrillation.

Dog 20. A large white mongrel female dog weighing 42 pounds was operated upon with the usual set-up of apparatus made and a constrictor placed about the right auricle. Constriction was applied for six 2 minute periods, which seemed to be long as this heart would go without dilatation with an onset of irregular rhythm and marked weakening of the force of contractions.

For each period it was thought that the time of constriction could be prolonged. Prompt recovery always followed. For the seventh period the constriction was carried for 3 minutes, in spite of dilatation and weakening. A prompt recovery followed. The next obstruction period was carried for 4 minutes, the last 20 seconds of which the heart dilated and ventricular contraction ceased. On release of the tape the heart greatly distended, the ventricles made slow feeble beats but after 30 seconds made no sign of recovery. One-half cubic centimeter of ephedrin in each ventricle was given and ventricular fibrillation immediately followed.

Dog 21. A small white male dog weighing 24 pounds, anesthetized with ether instead of nembutal, was used for this experiment. A No. 14 catheter was passed through the transverse sinus and then swung around the apex of the heart to act as a constrictor of the auricle. This heart did not tolerate constriction well and was carried for only 1½ minutes. At 1 minute and 15 seconds the heart dilated, rhythm slowed markedly and at 1 minute and 27 seconds the heart stopped beating. Prompt recovery followed, although the rhythm was somewhat irregular for 1 minute. After 3 minutes constriction was applied for 2 minutes with similar results. A 5 minute recovery period was allowed.

Constriction was applied for 3¼ minutes. The last 15 seconds the heart made only a few feeble beats and was widely dilated. On release the dilatation became extreme and the heart beat with weak flaccid strokes, was irregular for an additional 2 minutes. During this period the dog made irregular respiratory efforts, the effects of which made sharp rises in venous pressure. No massage or manipulation was made. When no sign of active recovery was seen, adrenalin hydrochloride 0.5 cubic centimeter was injected into each ventricle and a prompt recovery followed. The pressure rose to 94 millimeters. Between 1 minute and 45 seconds a 2:1 heart block was present.

With light ether anesthetic, the pupillary reflex was retained. On constriction the pupil contracted so at 30 seconds the pupils were pinpoint and then the pupils dilated so at the end of 1 minute they were widely dilated and remained so until release and recovery.

Two 3 minute constriction periods followed. The heart dilated each time before release, but recovered

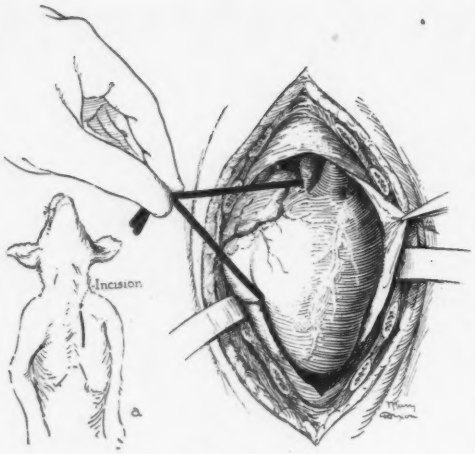

Fig. 9. Drawing showing the constrictor in place about the afferent vessels of the heart. When the circulation is obstructed normal cardiac contractions continue and the heart remains normal in size.

rather promptly after short periods of irregularity. The experiment was terminated.

DISCUSSION OF OBSERVATIONS

As referred to under "The Problem," the motive for undertaking this work was to see if a more efficacious method of constriction of the great vessels of the heart might not be determined. In answer to this question, it seems that the most important conclusion drawn from this experimental study is that it is possible to constrict the great vessels of the heart by a more satisfactory method than by any now in use.

This constrictor is so placed that all blood returning to the heart is cut off and this factor in itself provides many advantages. As was pointed out, some of the most obvious difficulties occurring when the pulmonary artery and aorta are obstructed, are caused by the great distention occurring in the heart. The heart exhausts itself rapidly laboring to overcome an impassable obstruction. In 3 dogs the pulmonary artery and aorta were constricted, one for the short period of 30 seconds and the 2 others for relatively short periods of 2 minutes and 40 seconds, and 3 minutes. In the latter 2 the dogs failed to survive and it seems logical to believe that some after-effect of distention is responsible for the failure of these hearts to recuperate.

The difference in the manner of recovery of the normal heart when these two methods of constriction are applied is certainly well illustrated on Dog 1, which was used for constriction by the older method, then 4 months later constricted by the new method, and reported under Dog 1A.

When the pulmonary artery and aorta were constricted for only 30 seconds the heart made a steady, slow recovery over a period of 35 seconds, making 70 beats; while following 3 minutes' constriction of the auricle, the same heart made a similar recovery in about 13 contractions and in 10 seconds.

In Dog 10, following constriction of the auricle for 2 minutes, the blood pressure reached a level high above normal in 9 contractions and 5 seconds. Compare Figures 4, 5, and 7.

There are apparently many factors which are combined to determine the final result of obstruction of these great vessels, but the most likely causes of cardiac damage are apparently two: (1) exhaustion of the myocardium; and (2) interference with the normal cardiac impulses and rhythm.

The death of 2 dogs of 3 used may have been only incidental, for it is too small a group from which to draw conclusions. However, as demonstrated by the experimental work of Laewen and Sievers which was performed on rabbits, it would seem that death was the rule for constrictions longer than 2½ minutes, and only a few animals survived for longer periods. These 2 animals followed the rule.

These dogs quickly developed signs of interference or interruption of impulses from the pace-maker. This may be due to any one of a number of causes or to a combination of these several effects. It is possibly from the muscular fatigue occasioned by the excess work put onto the heart at the time of obstruction. Lewis has demonstrated that:

> Heart block may be induced readily in cats, less readily in dogs by asphyxiating them. Such block is independent of the vagus occurring equally after full saturation with atropin or section of the vagi. It is also independent of blood pressure changes, of dilata-

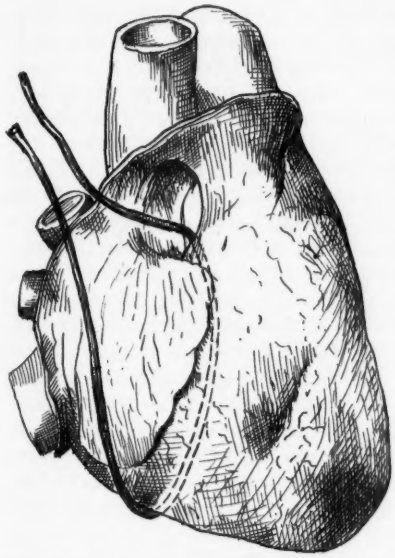

Fig. 10. The heart viewed from the right and in front, showing the constrictor in place about the afferent vessels through the great transverse sinus and as it crosses the right auricle.

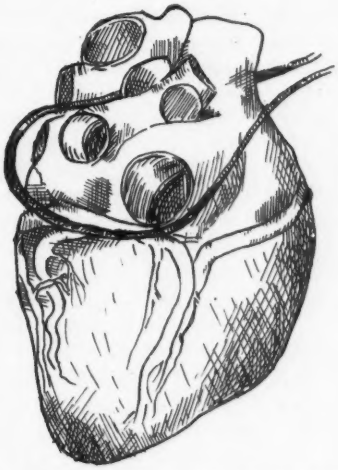

Fig. 11. Drawing of the heart viewed from below showing the constrictor in place about the afferent vessels as it passes through the great transverse sinus, under the two branches of the pulmonary artery, to go around the pulmonary veins and the inferior vena cava.

tion of the heart, and of excessive accumulation of carbon dioxide in the blood; it probably results from lack of oxygen and is perhaps due to an excess of acid products in the muscle.

That anoxemia plays a part in the development of cardiac arrhythmias or heart block, there seems little doubt and the excess work occasioned by obstruction of the pulmonary artery and aorta may be a factor in the production of anoxemia more quickly than when the afferent vessels of the heart are shut off and the heart has relatively no work to carry on. Certainly anoxemia promotes early musculature fatigue and excess work produces early anoxemia, so that a vicious cycle is set up.

The intracardiac pressure relations are difficult to evaluate in regard to their effect on the heart. This much may be safely stated, that when there is obstruction of the efferent vessels with a high intracardiac pressure, arrhythmias appear in some dogs as early as 1 minute (Dog 2) and most of the hearts became irregular as early as 1 minute and 30 seconds after constriction; while with obstruction of the afferent vessels and no increase in intracardiac tension irregularities do not usually appear until 2 minutes and 15 seconds, and many go 3 minutes, and one heart, Dog 17, went 5½ minutes with a regular rhythm during constriction. In general the maintenance of a regular cardiac rhythm during constriction seemed to be a fair index as to the length of time the obstruction could be maintained and have the animal recover.

We have thus outlined some of the advantages of obstructing the great heart vessels on the venous side, rather than on the arterial side. Because the heart may beat freely and not be overdistended and quickly exhausted, it is possible to interrupt circulation for longer periods than when constriction is applied to the pulmonary artery and aorta.

In all instances except on 3 dogs of 15 used, circulation was obstructed for periods of 3 minutes or longer with survival of the animals. These three exceptions are: Dog 19 was obstructed for 5 minutes and failed to survive; Dog 10 was obstructed for only 2 minutes and not constricted more after surviving; Dog 8 succumbed after 2 minutes' constriction. This was the first dog in which this method was

tried. In many instances it is difficult to tell just how long the constriction might be carried and still have the dog survive for they recovered several times when the obstruction was carried beyond what was considered a safe time. It seems reasonable to say, however, that the time of obstruction to the circulation by this method may be safely carried in the normal dog for a period of 2½ minutes. Although in all instances except those mentioned, constriction was carried beyond this time and in one instance was held as long as 6 minutes, this time of 2½ minutes seems to be the upper limit of safety.

The freedom from distention of the heart which this method allows and the continuation of the normal rhythm gives the advantage of a more rapid recovery of circulation, even with longer periods of interruption of the circulation. When the constrictor is released, there is a temporary dilatation of the right heart which is usually overcome in only a few heart beats and even during this time arterial circulation is going on, so that in these few heart beats the blood pressure reaches a normal level.

That there is a possibility of interference with the pace-maker when the constrictor is applied about the right auricle must be recognized so that when it is seen it may at once be relieved by a change of position of the constricting tube or tape. This phenomena was encountered in two instances in which a ventricular flutter came on with the application of the constrictor. Release and change of position in both instances corrected this condition and a normal rhythm followed. In Dog 11 this was seen with the second constriction period when the rhythm was very irregular and rapid, and when released because the heart was weakening so rapidly, the heart beat at a rate of 226 for 1½ minutes before it recovered. Again, in Dog 14, at the beginning of the third constriction period, the condition was seen and a change of position altered the condition.

When the heart is exposed to air and warmth, the drying action which occurs on the surface seems very rapid. It was necessary to apply some solution to keep the heart moist. The 2 solutions which were tried were physiological saline and Ringer's solution. Physiological salt solution was used first and seemed to have no effect other than merely to keep the heart moist. When warm Ringer's solution was applied to its surface, it was delightful to see in several instances a very marked stimulus to the heart. Although the sustaining qualities of Ringer's solution on the heart of the frog has been recognized for many years, it has not been mentioned in works dealing with cardiac surgery. It seems well at this time to call attention to the beneficial effects to be derived from the use of Ringer's or Locke's solutions when applied to the heart.

Ephedrin solution was used in two instances and both times caused immediate ventricular fibrillation (Dogs 19 and 20). Both instances were after prolonged and repeated constriction, respectively.

No dogmatic statements can be made in the conclusion as to the effects on the brain and vital medullary centers when the afferent vessels of the heart are constricted by this method. It seems, from the evidence presented by these animals in both the group which were kept alive following constriction and those done as acute experiments, that no serious damages to these vital centers could be demonstrated except following repeated, prolonged obstruction of the circulation.

In the animals which were kept alive in which either the superior vena cava or the auricle was constricted, there was no delay in the return of respiration in any dog and the following day the dogs seemed well oriented, friendly, and well. The cause of death seemed in no way related to the brain or vital centers, but to lung conditions. That dogs are not well suited to experimental work which involves opening of the pleural cavity has been demonstrated by Graham who has shown the presence of open interpleural sinuses in the mediastinal pleura. This means that one pleural cavity in the dog may not be invaded without the development of a bilateral pneumothorax, which is not tolerated.

It was possible to demonstrate clearly in 3 dogs in which it was tried that the respiratory center remained in a functional state after prolonged constriction. This could be

done for only brief intervals, nevertheless seemed satisfactory when by disconnecting the artificial respirator the dogs would soon make definite respiratory efforts. In Dog 11 this occurred after 14 minutes and after 21½ minutes of interrupted constriction, in Dogs 14 and 15 after 9½ minutes and 16½ minutes of interrupted constriction, respectively. In the other dogs this was not tried. That the vital centers will withstand such prolonged periods of obstructed circulation, some of the interrupted periods being 4 and 5 minutes of continuously blocked circulation, seems to support the contention that in this method of constriction the vital centers are protected by promoting a stasis of venous blood in the capillary bed from back pressure. It would be a problem of interest in itself to investigate the changes which occur in the brain when the various types of constriction are applied for interruption of the circulation. This might be done by direct observation by placing a window in the skull to observe the vascular changes which occur and by measuring the pressure changes which take place within the skull.

This method of constriction of the great vessels of the heart presents advantages which have heretofore not been available for human application. It only remains necessary to apply this technique which seems reasonable and entirely possible.

SUMMARY

1. A new method of constriction of the great vessels of the heart is described whereby the obstruction is placed on the afferent rather than on the efferent side of the heart.

2. It would seem that the constriction near the venæ cavæ lessens the likelihood of respiratory failure.

3. The time of constriction may be safely prolonged to 2½ minutes in the normal dog.

4. This method of constriction protects the normal cardiac conduction mechanism from the damages of cardiac distention.

I wish to express my sincere appreciation to Dr. Raymond W. McNealy, surgeon in chief of Wesley Memorial Hospital in Chicago, for his helpful suggestions and assistance in carrying on this work; and to Dr. Eugene Birchwood, who assisted in the experimental work in this problem.

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A RELIABLE METHOD FOR TESTING THE STERILITY OF SURGICAL CATGUT SUTURES

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THE object to be achieved in conducting bacteriological examinations of most substances is the demonstration of the presence or absence of some particular species of bacteria. However, the purpose of the bacteriological tests applied to surgical catgut sutures is quite different; for, in this case, the presence of any and all forms of bacteria is being sought. Such a procedure necessarily must involve a highly specialized technique carried out under the most rigid conditions, and must embrace a search for the spores and vegetative forms of all anaerobic as well as aerobic bacteria.

THE NEED FOR A RELIABLE TEST

After making extensive bacteriological tests over a period of years of catgut sutures manufactured in various countries, several experienced bacteriologists have reported a high percentage of non-sterility. In England, Bulloch found more than 75 per cent of the sutures of eight manufacturers to be non-sterile. In Germany, Knorr found that 80 per cent of commercial catgut sutures which he examined contained pathogenic and non-pathogenic bacteria. In the United States, Meleney and Chatfield found that the products of seven suture manufacturers contained living bacteria. In a 2½ year study (2) of the possibility of sterilizing catgut by chemical treatment, I found that copperized catgut sutures of one German manufacturer and three American manufacturers were infected with aerobic and anaerobic bacteria; also, that mercurialized catgut sutures of one American manufacturer were grossly contaminated. Subsequently, a 2 year investigation (3) by me of the sterility of foreign made catgut brought to light the fact that 62½ per cent of twenty-four brands were non-sterile; including the sutures of four British manufacturers, one French manufacturer, five German manufacturers, two Japanese manufacturers, and three Spanish manufacturers. In a survey (4) of the sterility of twelve brands of American made catgut conducted during a period of 5 consecutive years (1930–1934), I found that the products of six manufacturers were non-sterile repeatedly.

The large percentage of non-sterile catgut sutures found among the brands now marketed in the United States, as well as in foreign countries, emphasizes the need of rigidly testing the sterility of the sutures by a reliable method which embodies adequate controls. This subject is of vital importance to the surgical and hospital professions, for it has a direct bearing upon the welfare and life of surgical patients. Since the publication of my last paper (4) on catgut sterility, I have received numerous requests for a description of a reliable method for testing the sterility of catgut sutures; and it is hoped that the present paper will furnish the desired information.

IMPORTANCE OF CHEMICAL ANALYSES

As a result of their study of the sterility of catgut sutures, Meleney and Chatfield proposed a so called standard test (7) which they recommended for use by catgut firms and hospital laboratories. However, in applying this bacteriological test in connection with my study of chemically sterilized catgut sutures, I found (2) that the neutralizing solution was inadequate for removing large amounts of copper salts and mercury compounds with which some commercial sutures were found to be impregnated. During that research study, I showed the fallacy of depending upon any method for testing the sterility of catgut sutures unless the details of the chemical treatment to which they may have been subjected is known.

Therefore, before applying bacteriological tests to catgut sutures, I pointed out (2) that "*careful qualitative and quantitative chemical analyses of some of the sutures must first be made to ascertain the nature and amount of the chemical compound used to impregnate the*

sutures." Four sutures of any one lot are required for the purpose of making a complete chemical analysis. Then, a suitable neutralizing fluid must be devised and used to dissolve and remove the particular chemical substance found in the catgut sutures and which, if not removed, might inhibit bacterial growth. When applying bacteriological tests, it has been my custom to test not less than four sutures for the presence of aerobic bacteria and four other sutures of the same lot for the presence of anaerobic bacteria; thus using not less than eight sutures for the bacteriological tests. These eight sutures, together with the four required for chemical analyses, make a minimum of one dozen required for obtaining accurate information concerning the sterility of any one lot of sutures.

A complete set of controls is essential if the bacteriological test is to prove efficient and reliable. In addition to the controls which have been used and recommended by other bacteriologists when testing sutures for sterility, I have demonstrated (2) that three other controls are required if the test is to be adequately safeguarded. These will be included in the description of my technique.

A RELIABLE TEST

The bacteriological test outlined below is based primarily on the technique devised in 1926 by Dr. Benjamin White, and successfully used by him for several years for testing the sterility of catgut sutures. It also embodies the essentials of the method proposed by Meleney and Chatfield and that specified in the Therapeutic Substances Regulations of 1930 adopted by Great Britain.

The efficiency and the reliability of my test have been demonstrated over a period of 5 consecutive years (1930–1934), during which time it has been applied in testing the sterility of 12,522 surgical sutures; comprising twenty-four foreign brands, twelve American brands, as well as several thousand experimental sutures. During this time, the use of this test has successfully detected non-sterility of 62½ per cent of twenty-four foreign brands and 50 per cent of twelve American brands; and it has made it possible for me to demonstrate the fallacy of chemical sterilization of catgut sutures by revealing non-sterility of the 334 experimental lots which were subjected to the action of twenty-seven different chemical compounds under a wide variety of conditions.

Culture medium. The culture medium devised by Novy in 1893, and so successfully used by him since that time for anaerobic culture work, is used. This consists of bacteriological nutrient broth prepared by infusing one-half kilogram of beef for each liter of culture medium. To the broth are added 1 per cent peptone (Bacto "Difco"), ½ per cent sodium chloride, and 1 per cent glucose. To one-half of the medium (to be used for anaerobic tests) are added 2 per cent gelatin (Gold Medal) and 1/10 per cent litmus. The preliminary reaction is adjusted to a hydrogen-ion concentration of pH 7.6, so that after sterilization the final reaction will be pH 7.2–7.4. The medium is filtered and distributed in 40 cubic centimeter amounts into test tubes which are plugged with gauze-wrapped non-absorbent cotton and sterilized in the autoclave at twenty pounds pressure (125° C.) for 30 minutes.

A similar number of test tubes each containing 40 cubic centimeters of distilled water, and an equal number each containing 40 cubic centimeters of a neutralizing solution, are plugged and sterilized in the same manner.

Neutralizing fluids. These solutions are employed for dissolving and removing various chemical substances used in the treatment of the catgut itself or in the tubing fluid. Unless these chemicals are removed from the catgut so that their action is completely neutralized, they will be carried over into the culture medium where they will inhibit growth of bacteria that may be contained within the catgut. These fluids should comprise at least the following three formulas:

A. One per cent sodium thiosulphate and 1 per cent sodium carbonate in distilled water. This is the only neutralizing fluid required in testing the sterility of some brands of catgut sutures, and constitutes part of my regular technique. It will remove the usual chemical substances commonly found in tubing fluids and with which catgut may be impregnated—if not present in too great an amount.

B. Ten per cent solution of sodium thiosulphate in distilled water. This neutralizing fluid is required as a preliminary additional step in the technique if chemical analyses show that the sutures contain more than 2 per cent of a mercury compound or more than 5 per cent of iodine. In my research studies (2) on the chemical sterilization of catgut, I demonstrated that the large amount (3½ per cent) of a mercury compound, with which the sutures of one American manufacturer were impregnated, could not be removed with a neutralizing solution of 1 per cent sodium thiosulphate nor even with a 5 per cent solution; but that a 10 per cent solution of sodium thiosulphate would effectively remove all of the mercury compound.

C. Five per cent ammonium chloride in distilled water with ½ per cent ammonium hydroxide. This neutralizing fluid is required as a preliminary additional step in the regular technique if chemical analyses show that the sutures contain copper salts, even though present in very small amounts. Again, in my earlier research studies (2), I found that copper salts cannot be removed from catgut with neutralizing solutions containing either 1 per cent, 5 per cent, or 10 per cent sodium thiosulphate. After using many chemical solutions and making many tests, I found that copper salts can be entirely dissolved and removed from catgut by using the special neutralizing solution of ammonium chloride and ammonium hydroxide.

Anaerobiosis. The anaerobic seal originally devised by Hall is used because it effectively prevents evaporation as well as the re-absorption of oxygen. This seal consists of a mixture of equal parts of vaseline and paraffin ("vaspar") having a melting point of 53 degrees C., and is sterilized in the autoclave at twenty pounds pressure (125° C.) for a period of 1½ hours.

TECHNIQUE OF TEST

The exterior of the suture tubes is sterilized chemically by submerging the tubes for 24 hours in an active germicidal solution, such as ½ per cent potassium mercuric iodide. The tubes are then removed and placed between sterile towels.

The actual transfer of the sutures is made with a pair of obstetrical dressing forceps which has been boiled for one-half hour, then immersed in ethyl alcohol and thoroughly flamed. Between each transfer, the forceps is immersed in alcohol and thoroughly flamed.

Two operators wearing sterile caps, face masks, gowns, and rubber gloves work together and make transfers preferably in a dustproof, air conditioned chamber. After breaking the tube at the fracture mark, the strand of catgut is transferred to a test tube containing 40 cubic centimeters of sterile distilled water. As the transfer is being made, the cotton plug is removed from the test tube and the mouth of the tube is flamed. Then the tube is incubated for 24 hours. The purpose of this step is to permit the strand of catgut to absorb moisture and swell and partially untwist, thus permitting the neutralizing fluid in the next step to gain ready access to the interior of the strand.

Next, the strand of catgut is transferred to a test tube containing a sterile solution of 1 per cent sodium thiosulphate and 1 per cent sodium carbonate in distilled water, and is then incubated for 24 hours. The carbonate in this neutralizing fluid favors the continued swelling of the catgut, while the thiosulphate dissolves and removes the usual chemical substances, such as mercury, iodine, and chloramine (if not present in too great a quantity), with which the catgut may have been impregnated.

Then the catgut is transferred to a test tube of the culture medium, and those tubes which are to be incubated anaerobically are sealed with a layer 3 centimeters thick of the "vaspar" mixture. (The tubes of culture medium for the anaerobic tests are heated in the Arnold sterilizer at 100 degrees C. for at least 30 minutes to drive off oxygen, quickly cooled, promptly planted with the catgut sutures, and immediately sealed to prevent return of oxygen.) All culture medium tubes are incubated for 15 days and examined daily for growth.

Preliminary additional steps. If chemical analyses show that the sutures contain more than 2 per cent of a mercury compound or more than 5 per cent of iodine, the suture

strands to be tested for sterility must be transferred first to test tubes containing a sterile solution of 10 per cent sodium thiosulphate and then incubated for 24 hours. This special neutralizing fluid is used as a preliminary additional step to my regular bacteriological technique; so that the sodium thiosulphate will be removed from the sutures by the distilled water which constitutes the first regular step of the technique, and thus will not be carried over into the culture medium where it might inhibit bacterial growth.

Whenever chemical analyses reveal the presence of copper salts in the sutures, a special neutralizing fluid consisting of 5 per cent ammonium chloride with ½ per cent ammonium hydroxide must be used as a preliminary additional step to my regular bacteriological technique. The sutures should first be transferred aseptically to tubes containing this sterile neutralizing fluid and then incubated 24 hours. Then, they must again be transferred aseptically to tubes of this special neutralizing solution and again incubated 24 hours; so that the sutures will thus receive two treatments in the sterile ammonium chloride and ammonium hydroxide solution. Next, the sutures are put through the regular bacteriological technique already outlined.

Controls. 1. All culture medium tubes, immediately after sterilization, are incubated 3 days to bring out any gross contamination before being used in the test.

2. To prove the absolute sterility of the culture medium, a quantity of tubes not planted with catgut, equivalent to 2 to 5 per cent of the tubes of culture medium used, is incubated for the full length of the test.

3. The growth-promoting properties of the culture medium are controlled by a series of tubes planted with decimal dilutions of an active culture of Clostridium sporogenes, and growth should appear in the tube containing a dilution of 1:100,000 of the original culture; otherwise the culture medium is inferior for supporting bacterial growth.

4. In order to prove that chemical substances in sufficient strength to inhibit bacterial growth have not been carried over into the tubes of culture medium, the test organism (Clostridium sporogenes) is transplanted into tubes which have remained negative throughout the duration of the test.

In order to safeguard the test by eliminating the possibility of falsely positive results, the following *additional controls* are essential:

5. Two tubes of culture medium are planted with 10 cubic centimeters of each lot of distilled water and incubated for 15 days to determine the sterility of the solution.

6. Two tubes of culture medium are planted with 10 cubic centimeters of each lot of neutralizing solution used, and these tubes are incubated for 15 days to determine the sterility of the solution.

7. Two tubes of culture medium not planted with catgut but sealed with a layer 3 centimeters thick from every batch made of the "vaspar" mixture are incubated 15 days to determine the sterility of the anaerobic seal.

SUMMARY AND CONCLUSIONS

1. Before applying bacteriological tests to catgut sutures, complete chemical analyses of at least four sutures of any lot should be made to determine the nature and amount of the chemical compound with which the sutures may be impregnated.

2. Suitable neutralizing fluids must be devised and used to dissolve and remove the particular chemical substance found in the sutures, and which otherwise might inhibit bacterial growth. Three satisfactory neutralizing solutions for various chemicals are described.

3. Not less than eight sutures of the same lot should be tested bacteriologically if accurate information is desired concerning their sterility.

4. A reliable bacteriological test is described in detail; including the materials used, the actual steps of the technique, and a complete set of controls.

5. The efficiency and reliability of this test have been demonstrated over a period of 5 consecutive years (1930–1934); during which time it has been employed in testing the sterility of more than twelve thousand surgical sutures. The test has successfully detected non-sterility of 62½ per cent of twenty-four foreign brands and 50 per cent of twelve American brands; and it has revealed non-

sterility of 334 experimental lots of catgut sutures which were subjected to the action of twenty-seven different chemical compounds under a wide variety of conditions.

6. This reliable bacteriological test should be adopted by any authoritative body which may assume the obligation of controlling the sterility of catgut sutures in this country.

7. The Food and Drugs Administration of the United States Department of Agriculture has an adequately equipped bacteriological laboratory, a staff of bacteriologists, and a corps of collectors who could obtain the necessary sutures of all brands from the open market throughout the country. Here, therefore, the work of testing the sterility of catgut sutures could be satisfactorily conducted in an impartial manner. If an arrangement could be effected whereby this department of the Government would carry out the necessary bacteriological testing and report the results to the American College of Surgeons, publication of the results could then be made in SURGERY, GYNECOLOGY AND OBSTETRICS so that the surgical and hospital professions would know which brands of sutures are safe to use. Such a plan is earnestly recommended for serious and favorable consideration.

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PERIRENAL AND PERIPELVIC FIBROLIPOMATOSIS

Their Relation to Replacement Lipomatosis of the Kidney[1]

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In recent years numerous reports have appeared in the literature of cases of replacement lipomatosis of the kidney. This condition, which consists essentially of a replacement by hyperplastic adipose tissue of smaller or larger areas of the renal substance, is said to be very uncommon. Some authors (Hillman, Elansky) maintain that the hyperplasia of the perirenal and peripelvic fat is the primary pathological process and leads secondarily to pressure atrophy of the renal substance, while others (Young, Kretschmer) believe that the proliferation of the adipose tissue is secondary to chronic inflammatory changes in the renal tissue itself. The latter theory is supported by the fact that replacement lipomatosis is frequently associated with a calculous pyonephrosis or with a chronic suppurative process in the kidney. But cases are described in which histological examination of the renal tissue showed merely an "atrophy" and no sign whatever of any inflammatory changes. Such cases present a serious problem and have done much to arouse considerable speculation as to the true etiology of the process.

A study of each given case of replacement lipomatosis of the kidney reveals considerable destruction and shrinkage of the renal tissue, large areas of which seem to have been replaced by adipose tissue which shows a smaller or greater degree of fibrosis. Histological study of the renal tissue frequently shows low grade chronic inflammatory changes. Such a specimen obviously represents an end stage of pathological change. As in other forms of renal pathology, which lead eventually to considerable destruction and scarring of the renal tissue, a careful study of the final lesion gives no clue as to the nature, the sequence, or the severity of the various pathological processes which may have preceded. For these may long before have run their course and vanished, leaving in their wake only reparative changes in the form of a contracture and fibrosis, which represent the final lesion. An attempt to reconstruct the pathological development from a perusal of the individual specimen, is, therefore, apt to lead to false conclusions. In examining various forms of renal pathology on a large material, however, it becomes evident that certain cases resemble one another; and upon very careful scrutiny they prove to be various stages in the development of the same pathological process. If the cases prove to be sufficiently numerous and varied, it may become possible in this manner accurately to reconstruct the development of the lesion from its earliest to its latest stages.

We have followed this plan of attack in studying renal replacement lipomatosis.

SOURCE OF MATERIAL

In routine pathological examinations on a large surgical and postmortem material, several thousand kidneys, representing a great variety of renal pathology, were subjected to careful histological study. From these over a hundred cases, in which the perirenal and peripelvic fat showed significant changes, were selected for this study. In each case the topographical relation of the adipose tissue to the renal substance proper and the causal relation of the histological changes in the kidney to those in the adjacent fat were kept constantly in mind. Since the material examined is so large it will not be possible to give a detailed report of each individual case. It will, therefore, be the purpose of this paper to present the fundamental pathological changes as they occur in the various cases, to show their relationship to one another, and to trace their development from their onset to their end stages.

THE TOPOGRAPHY OF THE PERIRENAL AND PERIPELVIC FAT

In order accurately to follow the progressive changes in the perirenal and in the peripelvic

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Fig. 1. Peripelvic lipomatosis—in a case of chronic glomerulo-nephritis with renal contracture. The peripelvic fat shows a definite proliferation and gives the impression of having crowded into the kidney substance from the hilus. Autopsy specimen.

Fig. 2. Perirenal fibrolipomatosis—in a case of multiple purulent infarcts of the kidney. The fibrosed fatty capsule and the underlying fibrous capsule are firmly adherent to the surface of the kidney. Autopsy specimen.

fat, the topography of the renal substance and of the adjacent fat must be given due consideration. The kidney is covered by a paper thin transparent, fibrous capsule, or capsula propria, which is firmly attached to the renal tissue at the hilus. Elsewhere it may very easily be stripped from the kidney under normal conditions, leaving a smooth, glistening surface, with small, isolated bleeding points. The kidney, with its fibrous capsule, is surrounded by a mass of adipose tissue, or fatty capsule, which is several centimeters in thickness. This is divided into small lobules by fine, friable septa of connective tissue, which are loosely attached to the capsula propria. The fatty capsule is also attached loosely to the peritoneum on the one hand and to the retrorenal fascia on the other. Thus the fatty capsule serves not only to envelop the kidney but also to support it. Under normal conditions the fatty capsule has a loose, friable consistency so that the kidney, with its surrounding capsula propria, may very easily be shelled out of it by blunt dissection.

Surrounding the pelvis and the ureter, there is a thin layer of adipose tissue, which closely resembles the fatty capsule in its gross and microscopic structure, and extends in a thin sheath about the calyces to their point of attachment to the renal papillæ. The peripelvic fat also extends into the kidney as a thin layer surrounding the renal blood vessels and their ramifications. The fatty capsule (perirenal fat) and the peripelvic fat are more or less continuous with one another. In every case the perirenal and the peripelvic fat, as well as all projections of the latter, are sharply demarcated and separated from the kidney tissue proper.

LIPOMATOUS HYPERPLASIA

Various pathological conditions (benign nephrosclerosis, chronic glomerulonephritis, chronic pyelonephritis, and others) may eventually lead to a contracture of the kidney. The organ then shrinks leaving a rough, granular surface to which the fibrous capsule is more or less firmly adherent. The renal tissue also retracts from the hilus, leaving an appreciable space defect which is filled by a proliferation of the peripelvic fat. The latter then gives the impression of having crowded into the kidney from the hilus and of having replaced the shrunken renal tissue (Fig. 1). Careful gross and microscopic sectioning in every case, however, reveals that the hyperplastic adipose tissue is sharply demarcated from the renal tissue proper. Such a hyperplasia is a passive process rather than an active one. It is not

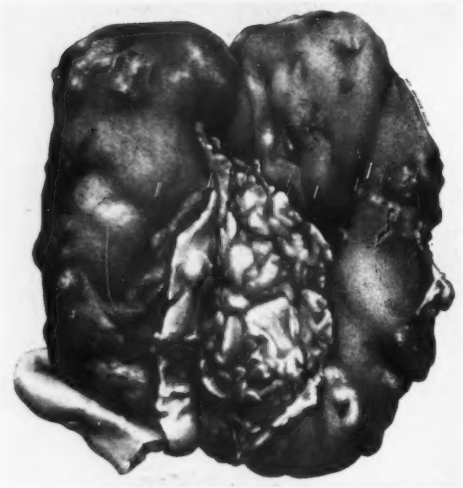

Fig. 3. Marked thickening of the capsula propria in a case of calculus pyelonephritis.

peculiar to the kidney and is not infrequently seen in other parts of the body, notably in the muscular tissues, when the latter undergo atrophy or shrinkage, and the fat surrounding the muscle sheaths undergoes a hyperplasia to fill the ensuing space defect (hyperplasia *ex vacuo*).

But the perirenal and the peripelvic fat may undergo an active hyperplasia under the stimulus of a low grade inflammatory process secondary to a chronic inflammation in the kidney.

FIBROLIPOMATOSIS—OR LIPOSCLEROSIS

In the presence of a low grade, chronic suppurative process in the kidney (chronic pyelonephritis, infected hydronephrosis, pyonephrosis), secondary inflammatory changes may appear in the perirenal and peripelvic fat which lead eventually to a fibrosis of the adipose tissue. In the early stages (which are encountered during pyelotomy for stone or nephrectomy for a mildly infected hydronephrosis), histological examination of the fatty capsule or of the peripelvic fat reveals low grade chronic inflammatory changes in the form of a small, round cell infiltration, especially in the fine fibrous septa between the fat lobules (Figs. 6 and 8). As the process advances the fibrous septa become thickened by an active proliferation of collagenic fibers and fibroblasts (Fig. 7). Gradually the connective tissue spreads into the lobules breaking them up into smaller areas (Fig. 8). Finally the entire fatty capsule becomes transformed into a firm fibrous sheath which surrounds the kidney like an armor plate (Fig. 9).

The sclerosed adipose tissue may then undergo a hyalinization which lends it a whitish, glistening appearance. Occasionally calcification or a metaplasia to hyaline cartilage or even to bone is also observed.

While these changes are progressing in the perirenal and peripelvic fat a similar fibrosis is simultaneously occurring in the capsula propria. The latter, which under normal conditions is as thin as tissue paper and transparent, gradually becomes more firm and opaque and may eventually reach a thickness of several mill meters (Fig. 3). At the same time the pronounced thickening of the fibrous septa of the fatty capsule, which attach the latter to the capsula propia, causes the sclerotic fatty capsule and the thickened capsula propia to become frozen into a firm fibrous mass. If it becomes necessary to extirpate such a kidney because of the underlying pathology (pyonephrosis) a plane of cleavage can be found only between the capsula propria and the surface of the kidney (intracapsular nephrectomy). The organ is then easily shelled out from the surrounding fibrous mantle until the hilus is reached. Since the capsula propria is firmly adherent to the renal tissue at this point, the capsule must be incised to expose the peripelvic fat which surrounds the renal vessels and the pelvis. After the peripelvic fat is pushed back, the clamps may usually be applied with ease and the kidney may be removed.

The sclerotic changes are usually more pronounced in the perirenal than in the peripelvic fat; and it is this fact which renders intracapsular nephrectomy a comparatively easy procedure in skilled hands in most cases. Occasionally, however, the sclerosis of the peripelvic fat is so marked that exposure of the pedicle can be accomplished only by sharp dissection. But fortunately this procedure is

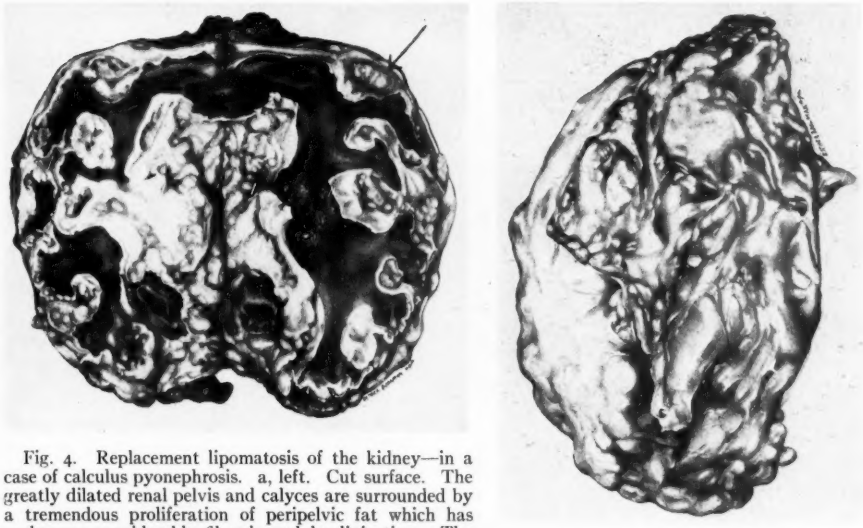

Fig. 4. Replacement lipomatosis of the kidney—in a case of calculus pyonephrosis. a, left. Cut surface. The greatly dilated renal pelvis and calyces are surrounded by a tremendous proliferation of peripelvic fat which has undergone considerable fibrosis and hyalinization. The contracted renal substance has been reduced to a mere shell (indicated by arrow) which covers the pathological adipose tissue like a capsule. b, Outer surface. The fibrosed fatty capsule and the thickened fibrous capsule are frozen into a solid fibrous sheath which is firmly adherent to the contracted shell of renal tissue. Removal of the kidney was accomplished only by extracapsular sharp dissection during which the vena cava was torn. Suture. Recovery.

not infrequently rendered less hazardous due to the fact that the pathological changes in the kidney have led to a severe renal contracture with a secondary shrinkage and obliteration of the renal blood vessels.

CHRONIC PYELONEPHRITIS WITH RENAL CONTRACTURE AND ITS RELATION TO FIBROLIPOMATOSIS AND REPLACEMENT LIPOMATOSIS IN VARIOUS SURGICAL CONDITIONS OF THE KIDNEY

Simple pyelonephritic contracture of the kidney. An acute pyelonephritis tends to heal because the copious secretions of the kidney continue to wash the infected material out of the renal tubules. But if the suppuration is maintained over longer periods of time by a constant reinfection from distant foci (prostate and seminal vesicles, intestinal tract, sinuses, etc.) or by a faulty drainage of the infected urine from the renal pelvis, a contracture of the kidney may eventually occur. The tubuli are first destroyed and disappear; and this is followed by an atrophy and hyalinization of the corresponding glomeruli (inactivity atrophy). Since a large part of the renal tissue is supplied with blood by the vasa efferentia of the glomeruli, a destruction of the latter produces a severe ischemia, which together with the secondary inflammatory changes in the interstitial tissue leads to a contracture and fibrosis of the kidney. The organ then shrinks, leaving a rough granular surface; and the renal tissue retracts markedly from the hilus. On cut section the renal substance manifests a definite narrowing, especially in the cortex. The cortical and medullary markings are usually indistinct. The peripelvic fat shows a definite proliferation and gives the impression of having crowded into the kidney from the hilus. A definite fibrosis is usually present in the fatty capsule and to a lesser degree in the peripelvic fat. Histological examination of such a kidney usually reveals definite traces of the preceding suppurative process, but, if the latter has run its course many months or years previously, one may find only numerous hyalinized glomeruli and a fibrosed interstitial tissue containing a few dilated tubules with a flattened epithelium

Fig. 5. Replacement lipomatosis of the kidney—in a case of calculus pyonephrosis. The greatly dilated pelvis and calyces are filled with pus and surrounded by a tremendous proliferation of peripelvic fat. The contracted renal tissue has been reduced to a paper thin shell which surrounds the entire mass like a capsule. The specimen weighed 9 pounds. Intracapsular nephrectomy.

and filled with coagulated colloid. It may then be very difficult indeed to identify such a contracture as having been caused by a previous chronic pyelonephritis (Fig. 10).

Primary pyonephrosis (small pyonephrosis). In the course of a chronic pyelonephritis, an obstruction to the outflow of the infected urine from the renal pelvis may develop. The stagnant urine then gradually becomes transformed into pus, leading to the formation of an empyema of the ureter, of the pelvis, and of the calyces above the point of obstruction. But since the preceding suppurative process has produced a sclerosis of the renal substance and a cellular infiltration of the wall of the renal pelvis and of the calyces, neither the pelvis nor the kidney tissue dilate following the development of the obstruction, and a small pyonephrosis is the end-result. Histologically such a kidney presents the picture of a chronic pyelonephritis with contracture.

Secondary pyonephrosis (infected hydronephrosis or large pyonephrosis). When an obstruction to the outflow of the urine from the renal pelvis (due to an aberrant vessel, a congenital valve, an abnormal insertion of the ureter, a renal or ureteral stone, etc.) appears before an infection has set in, a hydronephrosis develops first. The renal pelvis and the kidney substance may then dilate to a tremendous size; and, if an infection of the retained urine subsequently occurs, the latter is gradually converted into pus, and a large pyonephrosis results. Histological examination of the renal tissue in such a case reveals the changes of a chronic pyelonephritis as well as those of a hydronephrosis. Through the combined forces of hydronephrotic atrophy and pyelonephritic contracture, the renal substance is then reduced to a thin, fibrotic shell which stretches over the ends of the greatly elongated calyces like a huge canopy. This sequence of events leads to the formation of a large space defect which surrounds the renal pelvis and calyces, and which is bounded peripherally by the compressed shell of renal tissue. This space defect is filled by a proliferation of the peripelvic fat. The suppuration which is going on at the same time in the renal substance and in the renal pelvis gives rise to chronic secondary inflammatory changes in the hyperplastic peripelvic adipose tissue, which gradually lead to a fibrosis and frequently to a hyalinization of the latter (Fig. 4).

Such a kidney may reach an enormous size; and in extreme cases it may weight 10 pounds or more. If the organ is cut open it soon becomes evident that by far the greatest part consists of sclerotic adipose tissue which is pierced by the greatly dilated renal pelvis and calyces, and which is covered by a thin fibrotic shell of renal tissue, which surrounds it like a capsule (Fig. 5).

It is mainly such advanced cases which are known as a renal replacement lipomatosis.

RENAL REPLACEMENT LIPOMATOSIS

This condition consists of a hyperplasia and a sclerosis of the peripelvic fat associated with an advanced pyelonephritic contracture of the kidney. The inciting cause is always a low grade chronic suppurative process in the renal substance, usually of many years duration. The primary pathology is always the chronic suppuration in the renal tissue and the resulting renal contracture. The tremendous proliferation and fibrosis of the peripelvic adipose

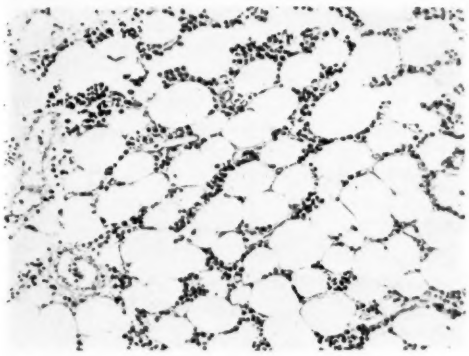

Fig. 6. Peripelvic fibrolipomatosis. High power photomicrograph showing chronic secondary inflammatory changes in the peripelvic fat.

tissue is always secondary to the changes in the kidney.

THE CLINICAL SIGNIFICANCE OF PERIRENAL FIBROLIPOMATOSIS AND OF RENAL REPLACEMENT LIPOMATOSIS

These two conditions assume clinical importance only from the fact that they produce extensive alterations in the topography of the tissues surrounding the kidney. The normal organ may very readily be shelled out by blunt dissection from its surrounding bed of adipose tissue, leaving a long narrow pedicle which may be clamped and tied with ease. As the result of pathological changes in the perirenal and peripelvic fat, however, considerable difficulty may be encountered. In the presence of a perirenal fibrolipomatosis the fibrous capsule is firmly adherent to the sclerotic fatty capsule. A plane of cleavage may then be found only between the fibrous capsule and the surface of the kidney; and the renal pedicle may be exposed only after sharp division of the reflected fibrous capsule at the hilus (intracapsular nephrectomy). In renal replacement lipomatosis (peripelvic fibrolipomatosis), a heavy band of fibrosed peripelvic adipose tissue surrounds the renal vessels like a mold. This produces a shortening and broadening of the pedicle. A preparation and narrowing of the latter for safe clamping and ligature can then be accomplished only by sharp dissection, which brings with it the danger of serious injury to the great vessels. It may truly be

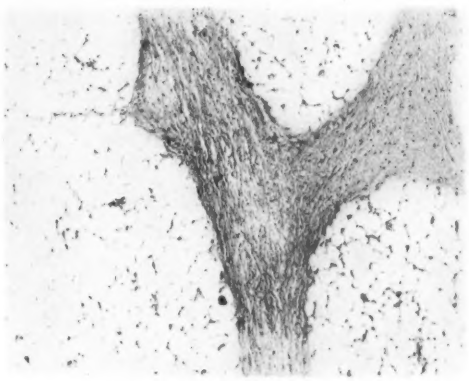

Fig. 7. Peripelvic fibrolipomatosis. Low power photomicrograph showing a thickening of the interlobular septa of the peripelvic fat.

said that if perirenal and peripelvic fibrolipomatosis did not exist, operations upon the kidney would be technically far simpler than they sometimes are.

Perirenal and peripelvic fibrolipomatosis are usually not recognized clinically. The diagnosis is commonly that of the underlying and causative pathological condition (pyonephrosis, or pyelonephritic contracture of the kidney).

PATHOLOGICAL DIFFERENTIAL DIAGNOSIS

Perirenal fibrolipomatosis and renal replacement lipomatosis are not infrequently con-

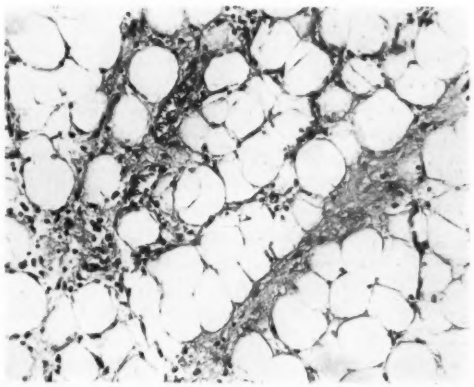

Fig. 8. Peripelvic fibrolipomatosis. High power photomicrograph showing fine strands of fibrous tissue spreading into the fat lobules and breaking them up into smaller areas.

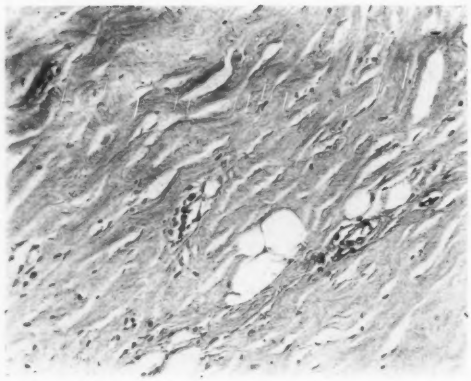

Fig. 9. Peripelvic fibrolipomatosis. High power photomicrograph showing an almost complete fibrosis of the peripelvic fat. Only a few scattered fat cells are still visible.

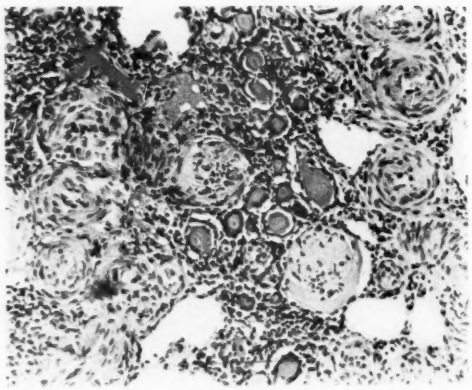

Fig. 10. Old pyelonephritic contracture of the kidney. Most of the glomeruli are completely hyalinized. The few remaining renal tubules are dilated and filled with coagulated colloid. The blood vessels are thickened and show degenerative changes. Active inflammatory changes are no longer demonstrable. Low power photomicrograph.

fused with various neoplasms of the perirenal and peripelvic fat, of the fibrous capsule, and of the renal substance proper. It is significant that such tumors may reach enormous proportions without producing any appreciable pressure atrophy of the renal tissue. Histological examination, therefore, reveals a perfectly normal kidney substance. In perirenal fibrolipomatosis and in renal replacement lipomatosis, on the other hand, a pyelonephritic contracture is always demonstrable; and one usually also finds traces of the preceding suppurative process in the kidney.

Simple lipomata of the kidney are usually small and situated in the cortex. They are well encapsulated and separated from the peripelvic fat by a heavy wall of normal renal tissue. Occasionally, however, such a growth may reach such a size that it occupies an entire pole of the kidney. The calyces of the involved portion then seem to end blindly in the tumor tissue. But in such a case careful gross and microscopic sectioning will reveal that the neoplasm is well encapsulated and sharply demarcated from the peripelvic fat.

If the growth of the above mentioned tumors is infiltrative, some confusion may arise on gross examination. But histological study will immediately reveal the blastomatous nature of the process.

In various forms of nephrosis the existing lipemia produces an infiltration of the renal tissue (mainly of the proximal convoluted tubules) with chemical fat. This lends the organ a yellow color. This condition, which is sometimes spoken of as a liposis, should never be confused with lipomatosis, which represents an extrarenal proliferation of actual adipose tissue.

SUMMARY AND CONCLUSIONS

It is generally assumed that replacement lipomatosis of the kidney is a rare condition. But comparative pathological studies show that it is merely an advanced form of peripelvic fibrolipomatosis, which accompanies every chronic suppurative process in the kidney to a lesser or greater degree. This latter condition represents a combination of a hyperplasia and a fibrosis of the peripelvic fat, occurring in response to a chronic suppuration and contracture of the kidney. In its advanced form the peripelvic fat gives the erroneous impression of having undergone a tremendous *active* proliferation and of having crowded into the renal substance, producing a pressure atrophy and destruction of the latter. Actually the changes in the peripelvic fat are secondary to those in the renal substance proper. Careful histological examination of the renal tissue in such cases will usually reveal a pyelonephritic contracture and traces of the preceding sup-

purative process (pyelonephritis). Occasionally, however, if the latter has run its course many months or years previously, no signs of any previous inflammatory change may be demonstrable, and it may then be very difficult to recognize the changes in the renal tissue as being definitely due to a previous suppurative process. Such conditions are especially apt to be encountered in the most advanced cases of replacement lipomatosis; and it is this fact which has led to the erroneous assumption by some authors that the changes in the kidney are those of a pressure atrophy due to an active overgrowth of the peripelvic fat.

All portions of the pathological adipose tissue in renal replacement lipomatosis are directly continuous with the rest of the peripelvic fat, and they are sharply demarcated from the renal tissue proper.

The development of a renal replacement lipomatosis depends upon the presence in the kidney of a low grade, chronic, suppurative process over a period of years. The circumstances which tend to maintain such a process are best encountered in cases of long continued incomplete obstruction to the outflow of the urine from the renal pelvis. This explains why renal replacement lipomatosis is so frequently seen in cases of renal stone.

Renal replacement lipomatosis must be sharply differentiated from neoplasms of the perirenal and peripelvic fat, of the fibrous capsule of the kidney, and of the renal substance. It should not be confused with liposis, which is an entirely different pathologic entity.

Perirenal fibrolipomatosis and renal replacement lipomatosis (advanced form of peripelvic fibrolipomatosis) assume clinical importance only from the fact that they produce very extensive alterations in the topography of the tissues surrounding the kidney. A detailed knowledge of these changes is essential to the proper performance of operations on the kidney.

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CLINICAL SURGERY

A SIMPLE SEVEN SUTURE METHOD OF BILATERAL URETERO-INTESTINAL IMPLANTATION

REPORT OF 12 CASES

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THE method, the surgeon, and the patient contribute in particular ways to the success of uretero-intestinal implantation. Many different methods have been used. Some of these are inherently defective and have shown a high percentage of failures no matter how skillfully performed. Others, which theoretically fulfill the requirements, frequently have failed because they were poorly executed. All methods are technical and require minute attention to every detail, and some are so technical as to defeat their purpose. A perfect operation may be followed by complications arising through individual predisposition to infection, formation of stone, or other abnormality. The age and condition of the patient affect the risk and the permanence of the efficient implantation. Young people have a better expectancy and fewer diatheses than adults and the aged. The results of uretero-intestinal implantation for exstrophy and vesicovaginal fistula surpass those for vesical cancer and tuberculosis. For example: In 1924, the right and left ureters of L. P., 5 years of age, were implanted into the bowel separately (September and November) and the exstrophic bladder was removed later. The result has been perfect (Fig. 1). Two years later, C. R. at the age of 6 years was treated similarly with a lasting result equally good (Fig. 2). However, M. R.,[1] 33 years of age, who had lived comfortably for 4½ years after implantation, died of uremia caused by the obstruction of a stone caught in the implanted ureter of her remaining kidney—a late complication unrelated to the method of implantation.

Whereas the technique of implantation is the important part of the problem of method, the preparation and after-care of the bowel and urinary tract are equally important and special. Good abdominal relaxation is essential and an abdomen full of adhesions as the result of previous surgery presents real difficulties. The technical requirements of any method may be stated as preventive. The prevention of peritonitis, urinary leakage, ureteral obstruction, and ascending urinary infection occupies a place of variable importance in different surgical minds, as shown by the variation in their methods of prevention. The late R. C. Coffey's efforts to avoid complications are evidenced by the idea of a functioning valve (submucosal implantation) at the point of implant, to prevent ascending infection, initial catheter drainage of the ureter to prevent obstruction from edema, abdominal packs to prevent peritonitis and take care of possible urinary leakage, and, later, the buried uretero-intestinal suture ("third technique") for the same purpose, obviating the need of packs but obstructing the ureter until the suture forms an orifice. Higgins, using Coffey's technique III, imbedded the ureter, without dividing it, in the wall of the bowel with the idea of securing free drainage of urine through the natural channel until the local edema subsided and the new orifice became established by the suture cutting its way through. Poth modified the Higgins' technique by discarding the uretero-intestinal suture (lumen to lumen), which was not strictly aseptic, for a second stage operation at which an orifice is formed between the intact ureter and bowel by a special cautery.[2]

[1] A right tuberculous kidney was removed in 1913, followed by a progressive tuberculous cystitis. In 1921, the capacity of the bladder was only 30 cubic centimeters and the total phthalein 25 per cent. The patient weighed 90 pounds and was miserable. Function improved to 55 per cent with an indwelling catheter but the left ureter never could be catheterized. Left uretero-intestinal implantation was performed in 1921. The ureter was enlarged and dilated. No tuberculosis was found at biopsy. The after-result was satisfactory. The patient weighed 121½ pounds within a year. She was brought to the hospital on July 6, 1925, in uremia. A skiagram showed a shadow in the region of the left ureter. Ureterolithotomy failed to relieve the anuria. At autopsy multiple acute abscesses were found in the left kidney, but there was no evidence of tuberculosis. The bladder was shrunken and almost healed. Chronic passive congestion of the lungs, fibrous myocarditis, acute splenic tumor, and focal necrosis of the liver were found.

[2] The ureter is divided distal to the part which has been imbedded in the wall of the bowel at a previous operation. A special cautery wire is passed through the cut end, in the same manner as a catheter, to the point at which the orifice is desired. An assistant meanwhile has passed a proctoscope to the intestinal side of this point. The current is turned on and the wire cuts through adjacent ureteral andi ntestinal walls, thus establishing an orifice. The assistant seizes the wire and draws it out through the proctoscope after it has been detached above by the surgeon. The loose ureteral end then is tied and covered with peritoneum or imbedded.

Fig. 1. Excretory urogram taken 15 years after ureterointestinal implantation for exstrophy. Both kidneys and ureters appear normal.

Fig. 2. Excretory urogram taken 13 years after ureterointestinal implantation for exstrophy. Renal function is normal and there has been no evidence of infection.

It is possible that the factor most responsible for either good or bad results by any method, once peritonitis and local infection are conquered, is the manner of the actual implantation. The more recent methods, some of which are heretofore mentioned, are planned mainly to prevent local infection and peritonitis. Undoubtedly, such infections are grave risks. However, I feel that their prevention has been emphasized at the expense of other equally important requirements. In looking back, I found that my own failures and successes could not always be explained satisfactorily and this review led me to suspect not only that the actual manner of implantation is more responsible for the good results than the method used, but also that a very marked simplification of method might lead to general improvement of results.

Omitting the details of an experimental study, which will be reported elsewhere, it may be stated axiomatically: (1) that the ureter should be led into the bowel by the most direct and natural route, without twisting, kinking, interference by bands, blood vessels or folds of peritoneum, too much tension or undue looseness; (2) that the incision in the muscular layers of the bowel should be made at the accessible point, in line with the natural course of the ureter and should be of suitable size, not too long or too short, exposing the surface of the submucosa (not the mucosa) without entering or breaking the blood vessels; (3) that all anchoring sutures should pass through the strong connective tissue fibers of the submucosal layer of the wall of the bowel and the adventitial layer of the wall of the ureter without entering the lumen or even the mucosa of either; and (4) that, in closing the incision, the ureter must not be constricted in the slightest degree, and at the same time the closure must be secure enough to prevent leakage or herniation.

A very simple technique will fulfill these requirements. Certain details of the following method need refinement and further simplification. However, by it 12 patients have had both ureters implanted at one operation and the method which was used is reported as a step in the development of a technique which differs from others by its simplicity.

THE SIMPLE SEVEN SUTURE METHOD WITH PROBE AND CAUTERY

The method to be described may be designated as the simple seven suture method with probe and cautery, to distinguish it from a similar technique without the use of the probe, to be reported later. The steps of procedure are:

1. The preparation of the patient;
2. The opening of the abdomen and the packing off of the intestines;
3. The preparation of the peritoneal flaps and isolation of the right and left ureters;
4. The selection of suitable positions for the implantation and the placing of stay sutures to mark the lower of these positions;
5. The implantation of the ureter which is to be placed in the lower position, and then the implantation of the other ureter;

Fig. 3.

Fig. 4.

Fig. 3. Drawing of the formation of the right flap of peritoneum which will be used to cover the site of implantation of the right ureter into the pelvic colon. The ureter is being hooked from its extraperitoneal bed by a ligature carrier.

Fig. 5.

Fig. 4. Drawing of pelvic view to illustrate the method of isolation of the ureter. As much length as possible is desired. By traction on the rubber tape, the point of entrance into the bladder can be seen transperitoneally. Through a short incision at this point, the ureter can be exposed and pulled out.

Fig. 5. Drawing of the abdominal view of the isolation of the ureter. The dotted line on the ureter near the bladder shows the point of division. The two ends are tied, the clamps removed, and the short slit in the peritoneum is closed with catgut.

6. The covering of the sites of implantation with the respective flaps of peritoneum;

7. The closure of the abdomen without drainage.

The particulars of each step are important and therefore are given in detail.

Step 1. The preparation of the patient is very important. The following outline gives the principal points:

1. The patient should have a non-residue diet for at least 3 days preceding operation.

2. Urinary antiseptics should be given.

3. Urograms should be taken and the non-protein nitrogen of the blood should be determined.

4. Castor oil, 1 ounce, should be given 2 days before the operation.

Fig. 6. Drawing which illustrates the selection of the favorable site for implantation so that the ureter has a natural straight entrance into the bowel. The line for incision, indicated by broken line beneath the ureter, is marked by stay sutures.

5. Three soap suds enemas should be given: the first, early in the afternoon of the day preceding operation; the second, on the evening of the same day; the third, on the morning of operation. All of this third enema must be siphoned back.

6. No fluids should be taken after 6 a.m.

7. Breakfast should be omitted.

8. A retention catheter should be placed and left open during the operation in order that the bladder will be empty and out of the way.

Spinal anesthesia gives the best relaxation and can be reinforced with gas and oxygen or ether if necessary. Ether is better for children.

Step 2. Abdominal exposure. A mid-abdominal incision is made from the symphysis to alongside the umbilicus. The intestines are packed off with a rubber sheet reinforced with gauze packs. The abdomen should be relaxed and the bowel must

Fig. 7. Drawing to illustrate the method of making the incision in the wall of the bowel down to the submucosa. The muscular layers can be separated from each other and stripped from the submucosa beneath by spreading open the points of a clamp in the incision.

be empty. Intestinal clamps then are unnecessary.

Step 3. Formation of peritoneal flaps and isolation of the ureters. The peritoneum is slit as near the pelvic colon as possible, alongside that portion into which the ureter is to be implanted, and not over the ureter. This incision is extended with scissors in both directions parallel to the gut. The outer layer then is stripped up, carefully so as not to tear it, beyond the position of the ureter both below and above the brim of the pelvis, and this flap of peritoneum is made of a sufficient size so that its edge can be drawn well out to the middle of the pelvic colon. The ureter strips up with this peritoneal flap as a rule (except in old people) and can be drawn out of its extraperitoneal bed by hooking it with a ligature carrier thrust beneath the peritoneal flap (Fig. 3). A tape of soft rubber is looped over the ureter which is dissected free of the peritoneum forming the flap, care being taken to interfere as little as possible with the peri-ureteral fascia and blood vessels and not to pinch, twist, or traumatize the ureter. By gentle traction on the tape, after the bladder has been lifted up into the suprapubic wound, the juxta-

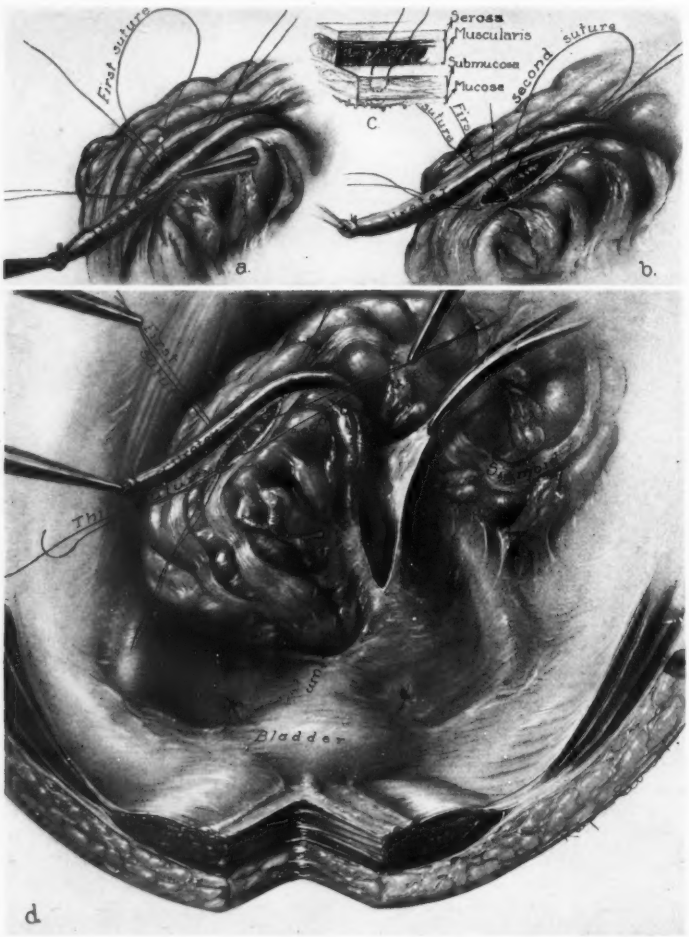

Fig. 8. Drawing to illustrate the manner in which the three anchoring sutures are placed. a, Suture, No. 1, passed from left to right through the submucosa, right to left through the adventitia, care being used not to enter the lumen of the bowel or ureter. b, Suture, No. 2, is passed opposite No. 1, keeping in mind the principles illustrated in Figure 10. c, Shows how each suture is placed in the submucosa. d, Shows the triangle of submucosa marked off by the three sutures.

vesical portion of the ureter can be seen transperitoneally. A short incision is made in the peritoneum at this point, and through this a small loop of the ureter is pulled out (Fig. 4). There the ureter is doubly clamped, divided between the clamps, and each end is ligated (Fig. 5). The short slit in the peritoneum next to the bladder, through which the ureter has been divided, is closed with catgut. By careful manipulation so as neither to traumatize nor to interfere with its blood supply, the freed and ligated end is pulled back and out from under the peritoneum. This leaves the ureter sufficiently long to enable transplantation at that point which later seems the most favorable.

A peritoneal flap is prepared and the ureter on the opposite side is isolated in a similar manner.

Step 4. Selection of site for implantation. After both ureters have been isolated, the anatomical relationships which have been created should be

studied. The most natural course of each ureter into the bowel must be determined in relation to the flaps of peritoneum on each side which have been prepared purposely to cover the sites of implantation and, if this relationship is wrong, the flaps should be modified to correct it by extending the incision in the peritoneum either up or down. It is preferable that the ureters should not enter the bowel at the same level and usually it is easier to place the left ureter higher than the right. This point can be settled by laying the ureters over the bowel and then manipulating ureters, bowel, and flaps into various positions until that which seems to be the most favorable is found.

Step 5. Implantation of the ureters with seven sutures. The position for implantation is marked by two stay sutures, one at each end of the imaginary line of incision (Fig. 6). The main purpose of these sutures is to mark the line for incision. They are used very little for traction. The part of the bowel marked by the sutures is held up and smoothed flat on the fingers of the left hand with the thumb. The sense of touch helps greatly in cutting to the depth desired and straight through the layers instead of on a slant or dissecting off between layers. A clean cut from 2.5 to 3 centimeters long (4 centimeters for hydro-ureter) is made in line with the two stay sutures through the muscular coats which are teased back by blunt dissection, thus exposing the surface of the submucosa with its blood vessels in the lower two-thirds of the incision for an area from 6 to 7 millimeters wide and 2 centimeters long (Fig. 7).

The ureter now is laid over this incision for suturing (Fig. 8). It has become distended with urine in the few minutes since ligation. This distention aids in making a neat implantation (natural course, proper length, more exact lateral suturing through the adventitia without perforation of the lumen, and more accurate judgment of the size of the triangle which will be formed on the submucosa by the three anchoring sutures). A suture of 20 day four o chromic gut on a one-half circle plastic atraumatic needle (D. & G.) is passed through the submucosa (care being taken that it does not enter the mucosa or the lumen of the bowel, Fig. 8, c) at the outer border of the area exposed, about 4 or 5 millimeters from its lower end, and then through the adventitia of the outer side of the ureter (care being taken that it does not enter the lumen or the mucosa even; flattening the ureter with the thumb on the index finger of the left hand aids in properly placing these sutures) at the proper point and side (Fig. 8, a) so that, when tied, a good fit will be secured. It is not tied at the time. The ends are left long

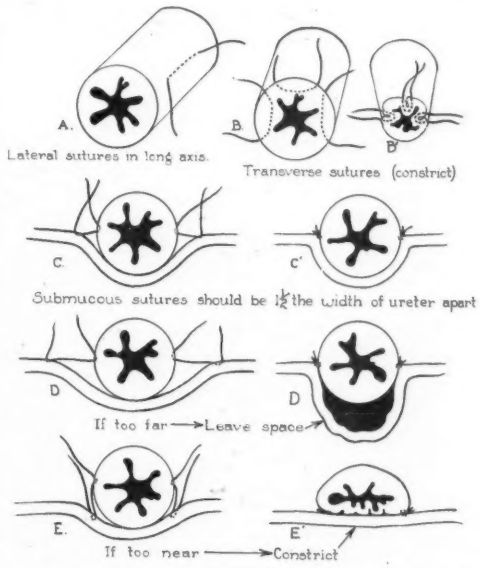

Fig. 9. Diagrammatic representation of the reasons for care in placing the anchoring sutures. A, The right method, a longitudinal suture. B, The wrong method, a transverse suture. B′, When tied, the transverse suture might constrict the ureter. C, The first and second sutures should be placed in the submucosa a distance apart of 1½ times the diameter of the ureter so that when tied the ureter fits snugly, as shown in C′, and not loosely when the sutures are too far apart, as shown in D and D′, nor too tightly when too close, as shown in E and E′.

and held in a clamp. A second suture is passed through the adventitia of the inner side of the ureter at a point just opposite the first suture (traction on the loop of the suture already placed in the ureter will help when the ureter is flattened out on the index finger with the thumb of the left hand) and then through the submucosa at the inner lower border of the area exposed, on the same level as but about 6 or 7 millimeters away from the first suture (Fig. 8, b). The distance between these first and second sutures should be one and one-half times the diameter of the ureter in diastole—distended (Fig. 9). The long ends of this second suture, which, like the first is not tied, are held in a clamp also. The third suture is placed through the submucosa at the lowest border of the area exposed and in the midline so that the three sutures form a triangle, the center of which is the point for perforation later (Fig. 8, d). This third suture is passed through the adventitia of the superior surface of the ureter in the proper relation to the two lateral sutures and

Fig. 10.

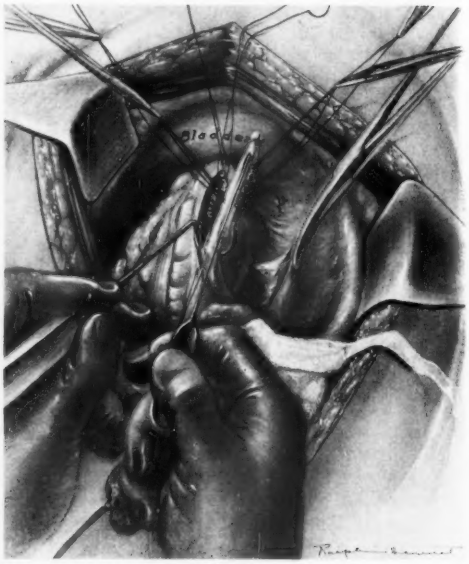

Fig. 11.

Fig. 10. Drawing which illustrates the cautery and probe method of inserting the ureter into the bowel. The cautery incision through submucosa and mucosa is made from the apex to just above the base of the triangle formed in the submucosa by the first, second, and third sutures.

Fig. 11. Drawing from another viewpoint to illustrate the method of inserting the ureter with the probe through the opening into the bowel made by the cautery. After the ureter is inserted, the mosquito clamps with rubber guards, which are not shown in the illustrations, on the loops of sutures No. 1, No. 2, and No. 3, are withdrawn and these sutures are drawn tight and tied, thus anchoring the ureter in position.

Fig. 12. Diagrammatic representation of the manner in which the ureter is anchored in the wall of the intestine by the five sutures. a, Shows how the sutures, Nos. 1, 2, and 3, anchor the ureter snugly in the small opening through the submucosa and mucosa made by the cautery and illustrates the reason this slit should not be too large but of a size, as indicated by the dotted circle, equal to the diameter of the ureter; b, represents the unconstricted anastomosis which is secured; and c, shows how the fourth and fifth sutures anchor the ureter in the trough of the incision.

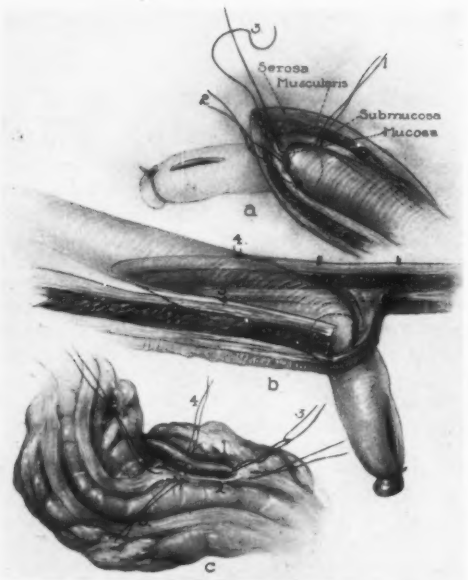

Fig. 12.

forms the third anchoring suture of the adventitia to the submucosa. The two parts of the loop of each of the three anchoring sutures which penetrate the submucosa are brought together and held in mosquito clamps fitted with rubber guards. Traction on these three clamps will lift the triangular area in the submucosa which is to be perforated. In this way the point for perforation and insertion of the ureter can be steadied when the time comes to implant the ureter, and the lifting of the wall of the bowel prevents contami-

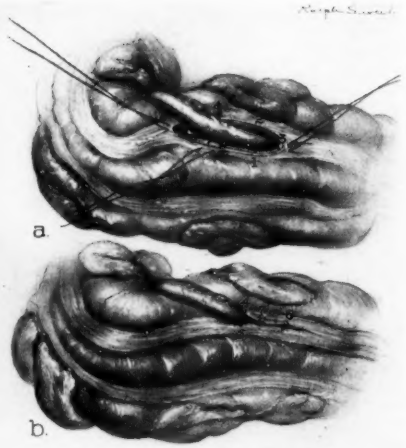

Fig. 13. Drawing to show how the sixth and seventh sutures are placed. Sutures 4 and 5 do not catch the submucosa since their purpose is to hold the ureter in the muscular trough. Suture 6 should catch a small strand of submucosa on each side, since otherwise it might tear out. Suture 7 closes the serous and muscular layers only. Catching the submucous layer in this suture might cause, when tied, constriction of the ureter and, since this suture is relieved of strain by suture 6, a submucous hold is unnecessary.

nation of the field by the regurgitation of any of its contents after it has been perforated. This will not occur if the bowel has been prepared properly and is empty. The distance between the three anchoring sutures in the submucosa is of some importance. The base of the isosocles triangle formed by them should approximate one and one-half times the diameter of the ureter so that when they are tied the ureter will fit the triangle. The sutures should pass lengthwise through the adventitia of the ureter, and not crosswise, in order that a good hold can be had without constricting the ureter when tied (Fig. 9).

An abdominal pad now is placed over the bowel and the ureter laid on it so that when the ureter is opened no urine can soil the wound. The tied end of the ureter should reach at least 3 centimeters beyond the level of the three anchoring sutures and it may be longer. This excess length will hang free in the bowel after implantation but will slough off after several days, either flush with the anastomosis or enough so that only a small papilla remains with the permanent orifice at its base. A temporary orifice beyond the wound and the point at which the new and permanent orifice will be, prevents urinary extravasation into the

Fig. 14. The method of extraperitonealization of the sites of implantation.

wound and makes the implantation easier from the technical standpoint. Ureteral obstruction from surgical edema during the first few days after operation also is less likely. About 1 centimeter from its ligated end, the ureter is slit so as to give a good, full, temporary opening (0.5 to 1 centimeter in size). A probe is passed through this slit into the blind end and given to an assistant who holds the ureter on the probe by pulling up the slack with a ligature carrier (Fig. 10). The gauze pad over the bowel is removed. The triangular area of submucosa is lifted taut with the three mosquito clamps, and a small opening is made with a fine cautery through it and the mucosa between the two lateral sutures. Care must be used to make this slit the size of the diameter of the ureter and not too long, as happened on one side in my last case when I used an electric knife. An incision which is too large gives a greater risk of leakage at the site of implantation.

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 15. Excretory urograms, taken at the time of discharge from the hospital, of the first patient operated on by this method (Case 1). The left renal pelvis and the right ureter appear dilated. The general condition of patient for 28 months since operation has been good (see Fig. 18).

Fig. 16. Exstrophy in a male 19 years of age (Case 2).

Fig. 17. Excretory urogram 2 months after total prostato-seminal vesiculocystectomy for cancer of the bladder with invasion of the prostate and 4 months after left hydro-uretero-intestinal implantation (Case 5). Function is reduced and the ureter is still dilated. However, it appears in better condition than 1 month previously (Fig. 22).

Fig. 18.

Fig. 19.

Fig. 20.

Fig. 18. Patient with an extensive epidermoid carcinoma of the neck of the bladder and whole urethra (Case 1) 10 months after vaginal cysto-urethrectomy (see Fig. 15).

Fig. 19. Photograph of the patient with the exstrophy shown in Figure 16 12 months after bilateral uretero-intestinal implantation, cystectomy, and plastic repair of the penis.

Fig. 20. Photograph of the patient with incurable vesicovaginal fistula (Case 9) 5 months after uretero-intestinal implantation (see Fig. 24).

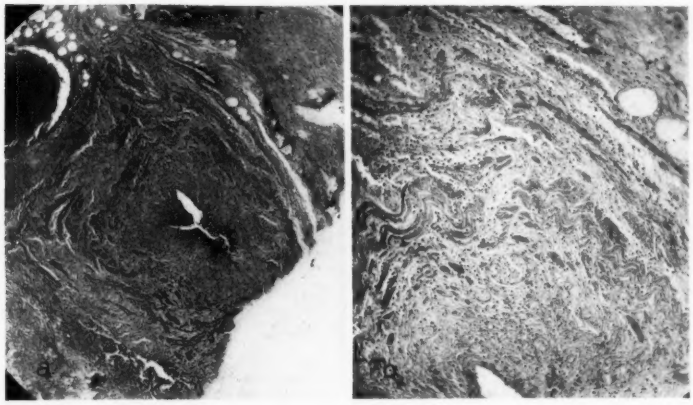

Fig. 21. Photomicrograph of a section of the ureter, of the 74 year old patient (Case 3) subjected to too much surgery, where it entered the lumen of the bowel showing periureteral inflammation and constriction. a, left, Low power; b, high power. The ureter and kidney above were normal.

The end of the ureter on the probe is guided into this opening by the assistant and pushed into the lumen of the bowel (Fig. 11). The three mosquito clamps are removed in succession and each time the corresponding anchoring suture is drawn tight. The withdrawal of the probe is the step in technique which involves the only real risk of sepsis and a modification of technique to avoid this is being tried out experimentally. After the probe has been withdrawn carefully onto a strip of gauze to avoid contamination of the area, these three sutures are tied.

Sutures No. 4 and No. 5 are used to anchor the ureter in the upper part of the muscular channel (1 to 1.5 centimeters in length) still left of the original incision. One of the cut edges of the incision is lifted in tissue forceps and a suture (suture No. 4) of the same material as that used for Nos. 1, 2, and 3 is passed through the serosa and muscularis of this lip without catching the

Fig. 22.

Fig. 23.

Fig. 24.

Fig. 22. Excretory urogram of the same patient as Figure 17, taken 1 month previously.

Fig. 23. Excretory urogram of Case 5, 12 months after hydro-uretero-intestinal implantation. The ureteral dilatation and the hydronephrosis are gradually disappearing.

Fig. 24. Excretory urograms taken 4 months after bilateral implantation of the ureters into the pelvic colon (Case 9). The kidneys and ureters appear normal.

TABULATION OF CASES*

Case Sex Age	Diagnosis and pathologic report	Operations	Course	Autopsy	Remarks
1 F 60	Carcinoma of the vesical neck, involving the entire urethra, with urinary obstruction ——— Epidermoid carcinoma; leucoplacia	1. Bilateral uretero-intestinal implantation (simple seven suture method). The opening in the bowel was made with scissors instead of cautery. Small wick drains were placed extraperitoneally to the site of the transplant on each side. Duration, 2 hours, 5 minutes ——— 2. Total vaginal cysto-urethrectomy 33 days after transplant	Stormy; temperature for 12 days. N.P.N., 61.8 on the tenth day, 33.2 on the twentieth day. There were signs of broncho-pneumonia on the eighth day which cleared up in 5 or 6 days. After the fifteenth day, recovery was good and rectal drainage was good ——— Very good. Patient discharged 53 days after transplantation. Figure 15 shows the excretory urograms at the time of discharge		The patient has been seen several times since operation. She has a small hernia in the lower end of the suprapubic wound. Photograph (Fig. 18) taken 10 months after operation
2 M. 19	Exstrophy of the bladder (Fig. 10)	1. Bilateral uretero-intestinal implantation (simple seven suture method, using probe and scissors). Extraperitoneal drains were used. Duration, 2 hours ——— 2. Cystectomy 3 months later	Uneventful; slight temperature for 8 days. The patient was discharged on the twenty-fourth day to return later for cystectomy ——— Uneventful		A good result. Photograph (Fig. 19) taken 12 months after operation
3 F. 74	Carcinoma of the urethra	Bilateral uretero-intestinal implantation (simple seven suture method used, except that the opening in the bowel was made with a pointed knife instead of a cautery). Cigarette drains were placed retroperitoneally. The patient was in poor condition at the end of the operation. The bladder was removed at the time	Fatal. The patient developed bronchopneumonia, arteriosclerotic heart disease with early congestive failure	Partial obstruction on the right with acute ureteritis and pyelitis. The left ureter and kidney look good but sections of that portion where the ureter enters the bowel (Fig. 21) show an inflammatory reaction. There are a paralytic ileus, myocardial hypertrophy and generalized arteriosclerosis, chronic adhesive pleurisy, and terminal bilateral bronchopneumonia	The operation took too long and the patient was in shock at the end. The technical steps were not done well and the operation was unsuccessful even if she had lived, as evidenced by the right sided obstruction. Removal of the bladder at the time of implantation of the ureters should not be attempted in an old or debilitated individual
4 M. 40	Tuberculous cystitis (advanced). Right nephrectomy for tuberculosis. Left nephrostomy for progressive hydronephrosis. (The kidney showed no evidence of tuberculosis)	The right ureter was implanted into the pelvic colon by simple seven suture method	Uneventful. A pyelogram taken through the nephrostomy tube shows that the uretero-intestinal anastomosis is patent. The nephrostomy wound healed quickly. The patient was discharged on the forty-seventh day		The patient was seen 20 months later. He had gained weight, was working and feeling well
5 M. 65	Carcinoma of the bladder with invasion of the prostate. (The patient's condition is very poor. Blood pressure, 210/95. No phthalein put out in 2 hours. Non-protein nitrogen, 163; creatinin, 4)	1. Bilateral nephrostomy with stormy convalescence but steady improvement later. The phthalein output was 55 per cent within a month, mostly from the left kidney. Blood pressure, 116/68 2. Left uretero-intestinal anastomosis. A hydroureter was found on the left side. The bowel was opened with a knife and not a cautery. The right hydro-ureter was doubly ligated but was not transplanted. Total prostatocystectomy. Penrose drains were placed extraperitoneally on each side	The patient made a remarkably good recovery		This patient recovered but the uretero-intestinal anastomosis of a hydroureter is not recommended. An excretory urogram taken 2 months after operation shows a poorly functioning left kidney (Figs. 17 and 22). Eight months later this kidney and ureter appeared more normal (Fig. 23). The ligation of a ureter to be left as a blind end for isolation of the kidney is not recommended. (See Case 7.) Nevertheless, this patient was getting along comfortably except for recurrent febrile attacks whenever the nephrostomy closed. He entered the hospital in November for right nephrectomy. No recurrence of cancer, a probable cure
6 M. 44	Tuberculous cystitis. Left ureteronephrectomy. Progressive hydronephrosis on the right	1. Left nephro-ureterectomy. 2. Right uretero-intestinal anastomosis (seven suture method)	Uneventful, except for abdominal distention the first 3 days after operation		A recent communication indicates that the patient's condition is excellent

TABULATION OF CASES*—Continued

Case Sex Age	Diagnosis and pathologic report	Operations	Course	Autospy	Remarks
7 M. 63	Squamous cell carcinoma of the bladder (extensive). Total phthalein, 55 per cent. Excretory urograms showed no dye on the left and a normal right ureter and renal pelvis	1. The left hydro-ureter was tied off but not transplanted. Right uretero-intestinal anastomosis. (The opening into the bowel was made with scissors and not with cautery) Convalescence was fair with periodic deviations of temperature 2. Total prostatocystectomy (7 weeks after transplant). Pathological examination showed extension into the prostate and left ureter. A small abscess was opened into extraperitoneally in the region of the ligation of the left ureter	Sudden exitus on the fifteenth day after cystectomy, 9 weeks after the transplantation	No autopsy. Impression: Suppurative left nephritis with perirenal and peri-ureteral abscesses. Apparently the transplant on the right was working satisfactorily	The patient died of sepsis secondary to the complete obstruction of the left ureter (see Case 5)
8 M. 64	Carcinoma of the prostate with extensive invasion of the bladder	Bilateral uretero-intestinal implantation. The opening in the bowel was made with Skene's cautery	The patient developed bronchopneumonia on both sides and died in 5 days	No autopsy	The operation technically was good
9 F. 42	Vesicovaginal fistula. Hysterectomy 4 years ago. An attempt to repair the fistula has been made each year since. Excretory urogram showed normal kidneys and ureters	Bilateral uretero-intestinal implantation, using Skene's cautery to open the bowel. There were very extensive intestinal adhesions which had to be freed before the field could be packed off and the pelvic colon exposed	There was considerable abdominal distention on the second day; urine was draining by rectal tube. The temperature rose to 104 degrees on the third day and was normal by the fifth day. The patient was passing gas and fecal material by the fifth day. Convalescence was uneventful from then on		The patient left the hospital in good condition and much pleased to be continent again. Figure 24 shows the excretory urograms and Figure 20 is a photograph of the patient
10 M. 63	Carcinoma of the bladder (biopsy), fulgurated four separate times without benefit. Blood pressure, 180/75	1. Bilateral uretero-intestinal implantation, using Skene's cautery to open the bowel. Duration, 1 hour and 30 minutes 2. Retroprostato-seminal vesiculocystectomy, October 27, 1934	Convalescence after uretero-intestinal transplantation was uneventful. Recovery after cystectomy was good. The patient was up on the eighth day, but developed septicemia from a pelvic abscess and died on the fifteenth day after cystectomy	Death from septicemia. Cortical abscess in left kidney without pyelitis or obstruction of ureters	Excellent result of transplants. (Fig. 25)
11 M. 72	Carcinoma of the prostate, vesicles, and bladder	1. Bilateral uretero-intestinal implantation, using Skene's cautery to open the bowel. 2. Cystectomy attempted. The pelvis was "frozen" and it was impossible to remove all of the cancer	Convalescence good after implantation. Suprapubic fecal fistula from injury of rectum when cystectomy was attempted. Patient died on the fifth day after cystectomy	The ureteral ends have not sloughed off (Fig. 26). There was no peritonitis or local infection	
12 F. 9	Complete incontinence following an automobile accident at the age of 3 years. Attempts to repair the incontinence were made at the age of 3 and again at the age of 6, without benefit. When the child was examined, no vagina could be found and a uterus was not palpable.† Apparently the vagina had been used to make a urethra. Ureteral implantation seemed the only way to secure continence. X-ray films show spina bifida of the entire sacrum and abnormal separation of the pubic bones of the symphysis. Intravenous urograms show normal kidneys and ureters	Bilateral uretero-intestinal implantation by the simple seven suture probe and cautery method. An electric knife was used and the opening made by it was too large on both sides, but more so on the right where an opening 1 cm. long occurred, necessitating suturing of the submucosa to close the bowel, a procedure, otherwise unnecessary	This patient had nausea and some vomiting with slight abdominal distention for 48 hours after operation. The highest temperature was 38.2 degrees and the temperature was normal on the fourth day. The blood urea on the second day was 28 mgm. per cent. The urine through the bowel was blood tinged for 48 hours but has been clear since	This patient has been seen several times since leaving the hospital and has remained in excellent condition	Figure 27 shows excretory urograms before and after operation Figure 28 is a photograph of the patient

*The analysis of other cases operated upon by the author by different methods than the one described in this paper is given in the paper entitled "Uretero-intestinal Implantation, etc.," Surg., Gynec & Obst., 1935, 60: 1115.

†At operation, ovaries and an infantile uterus were found but their relation to the bladder or vagina was undetermined.

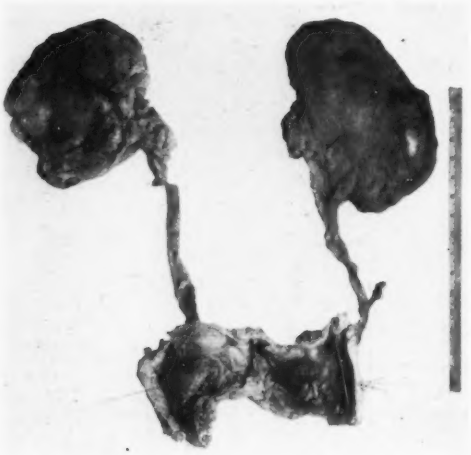

Fig. 25. Photograph of kidneys, ureters, and portion of pelvic colon, showing that the redundant ureteral ends have not sloughed off. The ureters and kidney pelves were dilated before operation because of obstruction from cancerous invasion of the bladder (Case 10).

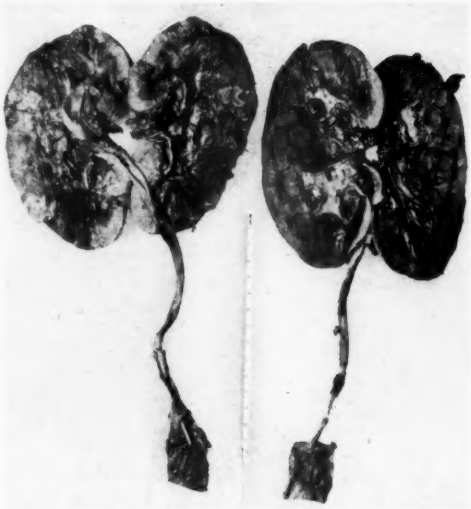

Fig. 26. The pelves and ureters are not dilated. The ureteral ends have sloughed off; the orifices into the bowel are well patent. Cortical abscess of the kidney, undoubtedly hematogenous in origin, is part of the picture of general septicemia (blood culture positive) resulting from abscess in the pelvis following cystectomy.

submucosa and then is passed longitudinally through the adventitia of the ureter at the proper lateral margin as it lies in the channel and back through muscularis and serosa, in the form of a mattress suture. Suture No. 5 is passed on the opposite side similarly through the other lip of the incision and through the adventitia of the adjacent side of the ureter. The surgeon must be absolutely certain that neither of these sutures perforates the ureter. When sutures Nos. 4 and 5 are tied, the ureter lies free and unobstructed but firmly imbedded in the muscular channel (Fig. 12).

Sutures No. 6 and No. 7 are of intestinal linen or silk (Pagenstecher). They close the lower portion of the incision (about 1 to 1.5 centimeters in length) by bringing together the cut edges of the serous and muscular layers of the bowel over the point at which the ureter perforates the submucosal and mucosal layers. The sixth suture is placed at the level of the third anchoring suture and should catch a small strand of submucosa on each side. The seventh suture closes the muscular layers of the bowel without submucosa just above the level of the first and second anchoring sutures (Fig. 13). If properly placed, they will prevent herniation of the bowel and will not cause obstruction of the ureter. Of course, more than two sutures may be used for this purpose if necessary, as is the case if too long an incision in the bowel has been made.

The other ureter is implanted by the same technique as that given above.

Step 6. Extraperitonealization. The flap of peritoneum on the right is adjusted to cover the area where the right ureter enters the bowel and is sewn in place with interrupted catgut sutures. The left side receives the same treatment. Care is taken that neither bowel nor ureter is pulled out of line by these flaps and that the direct and natural extraperitoneal course of each ureter is preserved (Fig. 14).

Step 7. Closure. The abdomen is closed in layers (peritoneum, fascia, connective tissue, skin) without drainage. It should be remembered that the bowel has been opened and there may have been some contamination of gloves and instruments. A complete change to a sterile set is necessary to insure against infection of the wound during closure.

REPORT OF CASES

Twelve patients have had simultaneous bilateral uretero-intestinal implantation by the simple seven suture method described above. (One had exstrophy; 2 had vesicovaginal fistula; 2 had tuberculous cystitis; 2 had cancer of the urethra and vesical neck; and 5 had cancer of the bladder.) Five patients have died. Only one of

Fig. 27, left. Excretory urograms before and after bilateral uretero-intestinal implantation (Case 12). a, November 18, 1934, 11 days before operation. b, December 14, 1934, 15 days after operation. This roentgenogram is of higher magnification than a; however, there is slight pelvic dilatation, particularly on the right.

Fig. 28. Photograph taken 2 weeks after a one stage bilateral uretero-intestinal anastomosis (Case 12).

the four deaths, however, occurred directly after the ureteral implantation (Case 8 in the table). This patient developed bilateral bronchopneumonia. Unfortunately an autopsy was not obtained. The ureters drained well from the beginning and the bowels were well open. There was no distention and no abdominal or renal tenderness—no indication clinically of any complication which might have arisen from a fault in surgical technique.

Three of the deaths (Cases 7, 10, and 11) followed an attempt to remove radically at a later time the bladder, prostate, and vesicles; one of these deaths, however, apparently can be attributed to renal sepsis arising from the ligation of a large hydro-ureter. It was thought safer at the time to tie it off than to implant it. Had nephrectomy been performed shortly after the cystectomy, this patient might have lived. There was no ascending infection on the implanted side. The other death (Case 3) was the result of too much surgery at one time; bilateral transplantation and removal of the bladder and urethra. Radical cystectomy for cancer carries as much risk as ureteral implantation. However, a risk of less than 10 per cent for the latter will fully justify its use in order to permit an attempt to remove a cancer which is incurable otherwise. All of the 5 patients who died had carcinoma which became operable only after deviation of the urine. Of the 7 patients living, 2 had cancer which was inoperable until after ureteral implantation. The time is too short to know whether these 2 patients have been cured of their cancer. At present both are well and show no evidence of recurrence after about 1 year. The 5 other patients (1 had exstrophy; 2 had tuberculous cystitis; and 2 had vesicovaginal fistula) are all doing well.

The main facts about the 12 patients are given in the table.

SUMMARY

The foregoing results, so far as the cure of cancer is concerned, are far from satisfactory. They are reported to illustrate a stage in the development of a technique of uretero-intestinal implantation which differs from others by its simplicity.[1] When one checks the requirements of the problem of transplantation with these early results, they give some encouragement. It is too early to form an opinion about late results.

In none of these twelve patients has there been any urinary or fecal leakage or sign of peritonitis. Infection at the site of transplant has not been found in those who have come to autopsy. There have been, however, surgical shock, 2 cases of bronchopneumonia, and some evidence of renal infection. There is room for improvement.

[1] The experimental work on dogs, in collaboration with Drs. Murphy, Wayman, and McCorkle, which led to the clinical trial of the simple seven suture method, will be discussed elsewhere.

ARTERIOVENOUS ANEURISM OF SUPERIOR THYROID ARTERY AND VEIN

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IN spite of the frequency of thyroidectomy, the occurrence of arteriovenous communications between the superior thyroid artery and vein is of great rarity. This is all the more remarkable, since the vessels are usually included in one ligature when the superior pole is ligated. In a similar anatomical situation, arteriovenous aneurisms are not uncommon in the stump of an extremity after amputation, when the artery and vein have been tied together. An interesting feature of these cases is the fact that the symptoms begin soon after operation and last for a great many years before the patient seeks relief. Because of the communication, these aneurisms rarely reach large sizes. Their chief symptom is an annoying bruit synchronous with the heart beat. This bruit is disturbing and frequently causes irritability, nervousness, and loss of sleep. They are not inherently dangerous, as they do not rupture, and they are too small to cause cardiac hypertrophy, so frequent in arteriovenous communications of large vessels. After operation, the relief from symptoms is immediate.

CASE 1. William A. Downes, in 1913, operated on a woman who had had a thyroidectomy 7 years before. She noticed a throbbing in the right side of her neck 6 months after operation. The swelling was the size of a goose egg. A diagnosis was made of an aneurism of the common carotid. The operation revealed a large communicating sac between the superior thyroid artery and vein. The artery and vein were ligated and the sac was easily removed. The patient made a satisfactory recovery.

CASE 2. J. M. Mora reported the case of a woman, aged 26, who had had a thyroidectomy 5 years before admission. A few days after operation she noticed a small swelling in the left side of the neck, which has persisted ever since. She also gave a history of syphilis. Her chief complaint was an annoying bruit, which disturbed her at night. There was a small expansile tumor, 5 centimeters in diameter, 2 centimeters below the mandible. A diagnosis was made of arteriovenous aneurism of the thyroid and vein. The aneurism was removed under local anesthesia. Recovery was uneventful.

CASE 3. Selman reported the case of a white female, aged 29, who was admitted with a complaint of edema, shortness of breath, palpitation, cough, and nervousness. She says that she has had heart trouble for 10 years. Four years before, a thyroidectomy was done. During the past year she has had a very severe unproductive cough. In spite of her symptoms, cardiac examination was comparatively normal. On the left side of the neck, in the region of the upper border of the thyroid cartilage, there was a small mass with a palpable thrill and a loud bruit. Under local anesthesia, the sac was dissected out. It proved to be an arteriovenous communication between the superior thyroid artery and vein. The interesting feature is that, although there was a single afferent artery, there were numerous veins, each emptying into the jugular. Following operation, the cough and shortness of breath subsided.

CASE 4. Author's case. E. L., aged 42 years, was admitted to the Jewish Hospital, November 2, 1934. She was referred by the neurological clinic with a complaint of nervousness and a loud disturbing noise in the neck. Thirteen years before, a thyroidectomy had been done. Following thyroidectomy she had aphonia for 3 months, which gradually disappeared. To the best of her knowledge, a swelling appeared in the neck a year after operation and

Fig. 1. Location of aneurism in author's case.

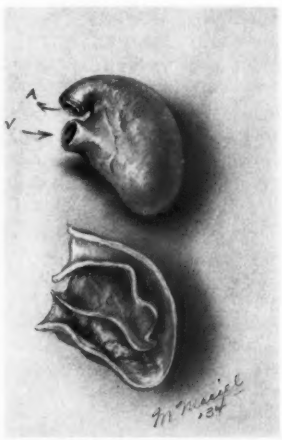

Fig. 2. Drawing of specimen.

had persisted ever since. Her chief complaint was the bruit, which kept her from sleeping.

General examination revealed a comparatively normal woman. Her blood pressure was 130/80, right arm; left arm, 134/80. Laryngoscopic examination showed a flaccid left cord. Wassermann reaction was negative. On the left side of the neck, mesial to the sternocleidomastoid muscle, at the left of the superior border of the thyroid cartilage, was an expansile swelling 2 centimeters in diameter. There was a loud bruit and a marked thrill. Pressure over the common carotid artery at Chassaignac's tubercle caused a cessation of the thrill. There was a scar, evidently of a thyroidectomy. Diagnosis of arteriovenous aneurism of the thyroid artery and vein was made.

Under local anesthesia, a longitudinal incision was made along the inner border of the sternocleidomastoid muscle. The sac was exposed, and there were found an efferent vessel leading directly from the external carotid and a single vein leading from the sac to the jugular vein. As can be seen from the drawing, the jugular vein was dilated to twice its normal size from the constant stream of arterial blood. The artery and vein were ligated with black silk and the sac easily removed. She left the hospital on the sixth day after operation.

The diagnosis in these cases is, of course, obvious. Their rarity may be estimated by the fact that in the different series of arteriovenous communications collected by Braman, Callander, Makins, and Reid, no mention of this condition is found.

An exhaustive search of the literature revealed the 3 cases herein reported.

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A NEW METHOD OF REDUCTION OF DISLOCATIONS AT THE SHOULDER JOINT[1]

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ON December 4, 1869, Theodore Kocher appeared before the Cantonal Medical Society at Berne and demonstrated on the cadaver his method of reduction of shoulder dislocation. Previous to this time the methods in vogue had relied chiefly upon abduction and traction in various combinations. Kocher presented his method as one employing finesse rather than force; making use of muscular spasm rather than combating it. It was the first thoughtful approach to the treatment of dislocation and has persisted to the present date as perhaps the one best method. Nevertheless, Kocher advised its use with certain limitations and very modestly and frankly stated that "in cases in which the head of the humerus fails to engage the glenoid fossa, other methods may be required." He believed the method contra-indicated (1) when the joint capsule was entirely torn off, especially when the upper and anterior portion of the capsule was separated; (2) when the humerus was fractured; (3) when the border of the glenoid fossa was broken. While few in number these contra-indications affect a large number of the cases of shoulder dislocation which must be treated.

In the remaining group of selected cases suitable to Kocher's maneuver, it has been my experience that considerable difficulty frequently arises in obtaining reduction. Either by lack of skill or knowledge on the part of the operator or by reason of the firmness of muscular and ligamentous attachments, it is often the case that the Kocher procedure requires several trials before success is attained. Even in the instances in which reduction is obtained promptly and easily, there is considerable trauma to the joint and its surrounding soft tissues by reason of the severity of the manipulation. For this reason I have attempted, as have many others, to fashion a satisfactory substitute. By reason of its loose capsular attachment and the shallowness of the glenoid fossa, the head of the humerus rather easily slips out of its normal position. Once dislocation has become complete, it is maintained not so much by bony contours and ligamentous engagement as by muscle spasm and, were the muscles removed, the head could be replaced quite as easily as it was dislocated. This can be very simply demonstrated on fresh autopsy material. As the position of the arm in which dislocation is most frequently incurred is one of abduction, it naturally follows that this position is the one in which dislocation is most easily reduced. Others, notably Stimson, have laid emphasis upon this feature and have devised various methods with it as a basis. Employing this basic principle of abduction and traction, which is not new, I have developed the following procedure which has proved uniformly satisfactory in my hands, and which I have reason to believe can be taught easily and applied in all cases.

If there is no fracture involving the joint and if the individual is not unusually muscular, anesthesia may not be necessary. As the manipulation does not increase the trauma or the degree of pain already present, it is my practice to make one attempt without anesthesia. In cases of fracture involving the glenoid or the humerus, no attempt at reduction should be made without complete anesthesia either local or general. The patient is placed on his back upon a table, the operator standing at his affected shoulder and facing toward his feet as in Figure 1. The affected arm is then taken by the wrist and with the elbow extended is abducted to 90 degrees. The elbow of the operator is then placed against the patient's chest wall just below the axilla as in Figure 2, and the palm of his hand engages the bend of the patient's elbow (Fig. 3). Using the base of his palm as a fulcrum, the operator then further flexes the patient's arm at the elbow joint, thus making use of a powerful lever which makes controlled traction upon the dislocated humerus. The operator completes the maneuver by still further flexing the arm at the elbow and adducting the humerus to 45 degrees as in Figure 4. At times it will be found that the operator's forearm is too short to provide an adequate leverage and in this circumstance a pad of the necessary thickness is introduced between his elbow and the chest wall of the patient (Fig. 5). By this means the operator's arm can be made to fit the dimensions of any patient.

The mechanical principles involved in this maneuver are extremely simple as may be seen from the accompanying line drawing (Fig. 6). The line *DF* represents the operator's forearm which rests against the patient's chest wall at *F* and supports the hand which acts as the fulcrum

[1]From the Department of Surgery, University of Minnesota and Minneapolis General Hospital.

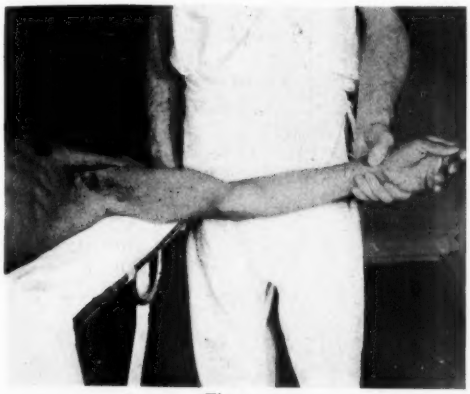

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

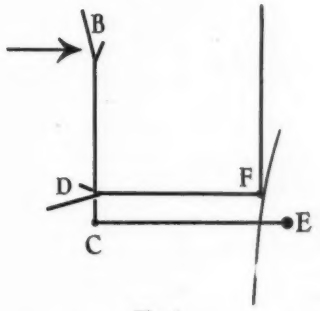

Fig. 6.

Figs. 1 to 5. Steps in method of reducing dislocations at shoulder joint.
Fig. 6. Diagram showing mechanics of procedure.

of a lever acting at the point *D*. The line *BC* represents the patient's forearm which acts as a lever with its power arm *BD* and weight arm *CD*. The line *CE* represents the dislocated humerus upon which traction is exerted by moving the lever *BC* in the direction indicated by the arrow. It will be seen that in this mechanism we have a

first class lever with the fulcrum at *D* and with a power arm *BD* and a weight arm *CD*. The pressure of the operator's hand against the point *B* (which is the patient's hand) in the direction of the arrow will result in traction upon the humerus *CE*. As the traction exerted upon the humerus is obtained through the action of a lever of the first class, with a relatively long power arm, the expenditure of only a small amount of force is necessary to effect powerful traction upon the dislocated humerus. Because the energy is so small in amount, the degree of traction on the humeral head can be very carefully and easily controlled.

For the past 4 or 5 years this method has been in use on the surgical service at the Minneapolis General Hospital and in my private practice. It has proved generally satisfactory and has not been attended by any unfortunate complications or results. I believe that this method has several advantages over those now in vogue.

1. It may be used in all cases of dislocation of the shoulder joint, even those complicated by fractures of the surgical neck, the tuberosity, or the shaft of the humerus or the glenoid fossa.

2. The maneuver may be employed without additional injury to the bone or soft tissue and, in fact, often times aids in the reduction of the accompanying fractures.

3. No force is applied to the dome of the axilla and consequently there is no danger of injury to vessels or nerve such as often occurs when the foot is placed in the axilla.

4. The method requires no great expenditure of strength and affords powerful controlled traction without the help of an assistant or special apparatus.

5. The method may be used in many cases without anesthesia.

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OSTEOMAS OF THE NASAL ACCESSORY SINUSES

With Report of a Case Illustrating the Transcranial Approach to Orbital Structures

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OSTEOMAS of the nasal accessory sinuses are microscopically benign tumors as they do not invade the surrounding tissues by cellular extension. They do, however, invade, destroy by pressure erosion, and displace neighboring structures by their gradual growth. The symptoms and signs produced by these osteomas depend upon their location and size. The only cure for them at the present time is complete surgical removal. When considered from this standpoint, they not infrequently present problems of considerable magnitude. In order to prevent complications which might result in death or to conserve the function of neighboring organs and prevent unsightly deformities, a careful study is required to determine their origin, structure, situation, and extent. Originating as they most commonly do in the nasal accessory sinuses, they may invade the cranial cavity, the orbit, the nasal cavity, the bones of the face, and other sinuses. If the tumor is so located that there is obstruction to drainage from the nose or accessory sinuses, secondary infection is likely to result and this may complicate both the clinical picture and the operative technique.

LOCATION AND FREQUENCY OF OCCURRENCE

Fetisoff (5) collected 258 cases of osteoma of the nose and sinuses. Those arising from the sinuses were distributed as follows:

Frontal sinuses, 131, or 50.7 per cent; ethmoidal labyrinth, 69, or 26.7 per cent; maxillary sinuses, 16, or 6.2 per cent; sphenoidal sinuses, 8, or 3.1 per cent. Thus osteomas originate more frequently in the frontal than in all the other paranasal sinuses. Many times these tumors involve more than one sinus and may be multiple themselves. They should be classified according to their point of origin rather than by the space they occupy or their direction of growth, but this site may be difficult to determine after growth has reached a large size.

ETIOLOGY AND PATHOLOGY

In another more recent article by Fetisoff (4) on the pathogenesis of osteomas of the nasal accessory sinuses, the usual microscopic classification of these tumors is followed, i.e., they are grouped, according to the type of osteoid tissue found, namely, eburnated, spongious, and mixed forms. After a review of the literature and a careful study of 4 cases, he arrives at the following conclusions: First, that the growth of the osteoma takes place from the inside outward by the metaplasia of fibrous tissue into bone tissue; second, that the osteoplastic ossificatory process takes place at the periphery of the osteoma and plays a secondary rôle in the growth of the tumor; third, that the fibrous tissue filling the interspaces of spongious bone is a direct continuation of the periosteum covering the osteoma from the outside; fourth, that the theory of origin of osteomas from the periosteum split off at the stage of the development of the organism is the most fundamental. This author believes also that the presence of split off periosteum at the early stage of development of the organism is not yet sufficient for the appearance of the tumor. He further states that, according to the recent theory of development of tumors, an exterior irritant is necessary, apart from the power of the cells, to produce tumor growth. Trauma and inflammatory processes in the nasal accessory sinuses may, undoubtedly, play the rôle of the exterior irritant in the development of an osteoma. There is evidence of this in the history of the majority of cases.

Grossly, the osteomas are usually irregular or nodular. They may be partially covered by the mucous membrane of the sinus in which they arise. There is also a periosteal covering. Many case reports emphasize the hardness of the bone and the difficulty which is encountered in attempting to remove it with the usual bone instruments. The spongious type, however, is easily excavated. The pedicle of attachment is usually small and easily separated. It may, however, be broad and firm.

AGE AND SEX

Adolf Eckert-Mobius reports the sex incidence in 180 cases of osteoma as follows: males, 110; females, 70. Of the age incidence given 102 of 163 cases were less than 30 years of age, while 61 were over 30. It is probable, however, that in many under 30 and especially those of greater age than 30, some had had the tumor a number of years. For instance, Cumstom states that cases are known

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Fig. 1. Mrs. J. Picture previous to operation showing the outward, downward, and forward displacement of the right eye.

of 15 or 20 years' duration and that he has knowledge of one which has been present for 42 years. Many cases are seen from puberty to the age of 20. These years cover the time in which the sinuses develop most rapidly and is most opportune for the development of osteomas.

SIZE AND RATE OF GROWTH

From the preceding paragraph, one realizes that the osteomas may be very slow in growth, their development usually being counted by years. It is also generally conceded that the growth is more rapid in younger individuals. The larger growths are usually found to be microscopically of the spongious type and are considered the more rapidly growing. The very hard, ivory-like tumors are usually smaller and are more slowly growing. Osteomas have been described as weighing as much as 1 pound but the larger ones are very rarely found in this age of surgery.

EXTENSION OF THE GROWTH AND RELATED SYMPTOMS

The orbit is by far the most common cavity attacked outside the sinuses themselves. The symptoms and signs produced by invasion of the orbit depend upon the degree of invasion and the location from which it takes place. Exophthalmos is the most common and prominent sign of osteoma of the sinuses with orbital invasion and, because a large percentage of these cases arise from the ethmoidal and frontal sinuses, the growth extends into the orbit from the superior or medial aspect or the angle between the superior and medial aspects. This results in displacement of the eye downward, outward, and forward. Should the invasion take place from the lateral or the inferior walls of the orbit, the displacement of the eye is medially and upward.

There may be considerable displacement of the eye without apparent or very little disturbance of vision. Pressure or tension on the optic nerve may interfere with vision. If pressure is exerted against the muscles or the nerves to the muscles, oculomotor palsies may result. If the circulation is interfered with, there may be an ischemia or papilledema of the retina depending upon whether the obstruction affects primarily the arterial supply or the return venous flow. Should the eye be pushed forward so far that the lids fail to cover the globe, corneal ulceration and its complications are likely to ensue. Also, inflammatory conditions in the nose and sinuses may find their way along the growth and cause a similar reaction in the orbital tissues.

In osteomas arising from the frontal and ethmoidal sinuses, if the direction of growth is upward and backward, erosion of the bony wall of the cranial fossa takes place and the tumor becomes partially intracranial. Displacement or perforation of the dura with pressure upon the frontal lobe may eventuate without symptoms as long as no complications occur. However, perforation may release cerebrospinal fluid which passes along the growth and drains by way of the sinuses and the nose. Infection may thus occur by this route and produce brain abscess or meningitis. Cushing reported 4 cases of osteoma, in one of which cerebrospinal rhinorrhea communicating with the cerebral ventricle took place; in a second, there was a huge intracerebral pneumatocele; while in a third there was found an intradural mucocele. Two of his cases were further complicated by infections and the patients died of meningitis and brain abscess.

The most frequent cause of disaster following the surgical removal or attempts at surgical removal of these tumors has been intracranial infection. This must be considered of prime importance when the cranial cavity has been entered and especially when the dura has been opened.

When osteomas invade the nasal cavity, the symptoms are those of obstruction, loss of smell and discharge. Obstruction frequently is complicated by infection due to improper drainage. If the growth is limited to the sinuses, in which no obstruction to drainage takes place, usually no symptoms occur. However, when obstruction is present a mucocele may develop, or infection may occur. The obvious symptoms are the result of these secondary changes. An osteoma may be found with X-ray on routine examination for

Fig. 2. Roentgenogram taken in the occipitofrontal position.

Fig. 3. Roentgenogram taken in the lateral position.

other conditions in which the tumor produces no symptoms whatsoever. If the growth is large enough, all the neighboring cavities may be affected by a single tumor, i.e., the frontal sinus, the ethmoids, the nasal cavity, the cranial cavity, and the orbits.

PAIN AND HEADACHE

Pain and headache are found only occasionally in the presence of osteomas. Many patients with large tumors have never been disturbed with either symptom, and it is probable that headache or pain does not occur except as follows: (1) when there is pressure on a sensory nerve, (2) when secondary infection is present, (3) when drainage from the sinuses has been obstructed, (4) when an intracranial complication has arisen.

DIAGNOSIS

The diagnosis of an osteoma of the sinuses, if of appreciable size so that it has displaced other organs, can scarcely be mistaken for other growths if an X-ray is taken. However, in 1 case Cushing mistook the lesion for an ossifying meningioma. It is possible for a growth to occur about the orbits in which bone formation or calcium deposits may occur. In the main, careful X-ray studies must be relied upon for diagnosis of the lesion.

PROGNOSIS

Among the reported cases in which patients were not operated upon, a mortality of 48 per cent due to complications is recorded (A. Eckert-Mobius). This, no doubt, is far too large, as only the more severe and complicated cases are of sufficient interest to report. The operative mortality has been estimated at from 7 to 10 per cent. The location and extent of the tumor is of the utmost importance in the prognosis. Small tumors located in the frontal, ethmoidal, and maxillary sinuses should be removed without difficulty and with practically no mortality, while tumors located in the sphenoids may be practically inaccessible and the danger of surgery may be so great that one is not justified in attempting removal. Tumors which invade the cranial cavity are the ones in which complications following operation have most frequently ended fatally.

SURGICAL CONSIDERATIONS

The only method that will produce a complete and permanent cure of the condition is the careful radical removal of the tumor, together with its pedicle and a portion of bone from which the pedicle is derived. The eburnated or sclerosing type may not recur when broken from its attachment, even though the pedicle remains. The mixed type and especially the spongious type is prone to recur if separated in this manner. Therefore, one should plan an operative procedure which will give sufficient exposure not only to remove the tumor together with its site of origin but will also

Fig. 4.

Fig. 5.

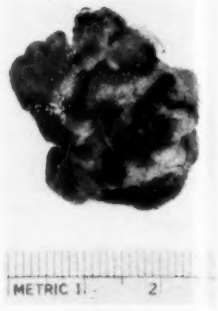

Fig. 7.

Fig. 4. Picture taken 9 days after operation showing some improvement in the displacement of the eye.

Fig. 5. Picture taken 10 days after operation showing the scar within the hairline and the site of the rubber tissue drain through the incision.

Fig. 7. Photograph of the tumor removed at operation.

enable one to deal with any complication that may arise from its position and location. This is especially true in the tumors that penetrate the cranial cavity. One should conserve, in so far as possible, the function of the neighboring organs, especially the eye. Also, it is desirable to prevent deformity and unsightly scars. It is obvious that where there is such a variation in size and position of the growth, each case will demand planning that is applicable to its peculiarities. In a large proportion of cases reported previously, the exposure has been either that which is used by ophthalmological surgeons for investigating the orbit or the one employed by rhinologists for external operations upon the paranasal sinuses, with such modifications as the particular tumor necessitated. In 1927, Cushing proposed a new method of exposing osteomas which projected into the cranial chambers, producing intracranial complications.

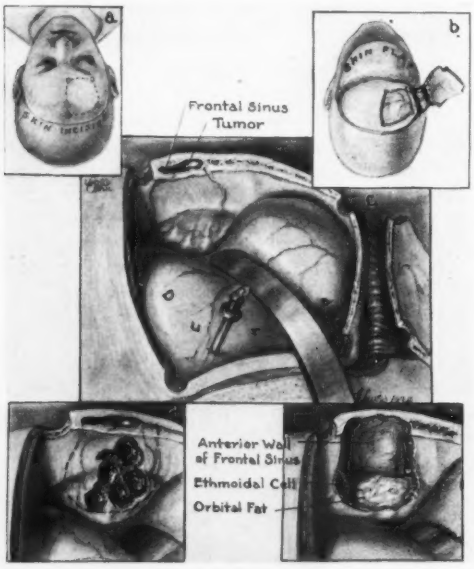

Fig. 6. a. The skin incision and location of the bone flap; b, the skin flap turned forward and the bone flap raised with pedicle of temporal muscle; c, detailed view showing the dura retracted from the posterior frontal sinus wall and the roof of the orbit, with the tumor in the frontal sinus outlined behind the wall of the sinus and the orbit. The needle is placed in the ventricle to release tension; d, the area excavated by the bone drill; e, the resulting cavity after the tumor was removed. The anterior wall of the frontal sinus is somewhat eroded by the growth, the orbital fat arose in the position formerly occupied by the tumor and an ethmoidal cell is uncapped. The nasal frontal duct is open.

INDICATION FOR OPERATION

Since there is great variation in the final result of cases in which operation has not been done, namely, a course which may proceed without symptoms or one which may terminate in death, it becomes necessary to determine the possible or probable course before deciding on the type of treatment. Hence, if a small eburnated, symptomless osteoma is discovered during the course of an examination by roentgenogram of the head and if present in a young individual, it might be checked up in from 4 to 6 months' time. If there is then noticeable an increase in size or in symptoms, operative treatment should be instituted. If this tumor occurs in an adult, a much longer time might elapse before checking it up with X-ray, for, as a rule, they grow much slower in an adult than in youth. They may never require treatment.

Should a similar tumor be found and be thought to be of the spongious or mixed type, due to its

poor calcification, it should be observed more frequently and again operative intervention should be considered upon increase in the size of the growth or if symptoms are produced by it.

When a patient presents himself with a well developed tumor which is causing deformity, exophthalmos, obstruction of the nose or nasal sinuses, with resulting infection or mucocele, or with an intracranial complication, the indication for operative treatment is already present and one need not delay other than to prepare the patient in such a manner as will render him a suitable operative risk.

Cushing's method of approach is that commonly used in operations upon the pituitary in which the anterior cranial fossa is entered over the roof of the orbit by turning down toward the temporal fossa a full thickness flap and retracting the dura. This gives a wide exposure of the roof of the orbit and dura overlying the ethmoidal and frontal sinuses, and will permit the repair of the dura by suture or the application of a fascia transplant. After the dura has been repaired the tumor can be attacked by removing the roof of the orbit or the orbital wall of the ethmoidal and frontal sinuses. This approach, which may be called a transcranial exposure of the orbit, besides giving an exposure through which the dura may be adequately repaired makes it possible to retain the supra-orbital arch of the frontal bone. This prevents deformity which would result from the removal of the bone which forms the arch. It is also possible to remove a large growth upward with less trauma to the orbital contents.

Naffziger's (6) operation for decompression of the orbits in progressive exophthalmos uses a similar approach. He employs a transverse incision above the hairline to hide any scarring. The scalp is turned forward and a bone flap made as in a transfrontal approach to the pituitary. When infection is present, the method of Cushing permits better drainage and the flap may be left open until the infection is cleared up and secondarily closed.

The transcranial approach to the orbit has seldom been described. It gives an excellent exposure from the orbital arch anteriorly to the sphenoidal ridge posteriorly and is applicable for exposing any tumor in the upper portion of the orbit as well as in the floor of the anterior cranial fossa. This approach was chosen in the case to be reported (1) because the attachment of tumor was evidently on the cranial wall of the orbit and frontal sinus; (2) because complete removal necessitated the exposure of dura; (3) because it was felt that there would be less disturbance of the orbital content; (4) because there would be no visible scar; and (5) because the orbital arch would be preserved, preventing deformity which would occur if the growth were attacked from the usual anterior approach. The latter would necessitate removal of the supra-orbital arch and anterior wall of the frontal sinus, leaving some scarring and make a plastic procedure necessary to correct the resulting deformity.

Fig. 8. Photograph which was taken 50 days following operation, at which time the hair had covered the scar of the original incision and the eyebrows had grown in. There is practically no ptosis of the right upper eyelid and the globe is not noticeably displaced.

CASE REPORT

The patient, a shoe factory worker, was a woman aged 20 years. She came to the Clinic on December 7, 1933, stating that she believed her eyes had been prominent for the last year. She felt nervous and tired easily. She had been told that her condition was due to goiter. Her family history was negative and her past history revealed good health on the whole although she had had a tonsillectomy for tonsillitis and an attack of abdominal pain for which an appendectomy had been performed. She had no other symptoms except an occasional headache and diplopia after prolonged use of her eyes or when she became fatigued. During the past year she felt that she had been more nervous, tired more easily, and was much more irritable than previously, but in spite of this she had gained 15 pounds in weight.

Her general physical examination revealed a well developed and nourished woman of 20 with a pulse rate of 96, regular in rhythm and a blood pressure of 120 systolic and 90 diastolic. The chest, including the heart and lungs, was found to be normal on examination. The same was true of the abdominal, the rectal, and the vaginal examinations as well as that of the extremities and of the reflexes. The special thyroid examination showed unilateral exophthalmos (Fig. 1), some nervousness and instability, but no other signs of toxicity, while her thyroid gland was normal in size and consistency. The ears, nose, and throat were

quite normal with the exception of a mild subsiding rhinitis.

The laboratory report revealed a negative blood Wassermann and the blood counts and urine analysis were within normal limits. The examination of the eyes showed the right globe to be displaced forward, laterally, and downward. There was good function of all the muscles in spite of the displacement and there was diplopia only when the muscles were fatigued. There was moderate ptosis of the right upper lid. The ophthalmoscope disclosed no abnormality of the fundi. Palpation under the right orbital ridge revealed an irregular, immovable tumor of bony hardness. X-rays of this area showed an osteoma of the frontal sinus apparently originating on the cranial wall of the sinus and the orbit. This tumor extended forward and downward into the orbit and frontal sinus, and also upward within the sinus. The size and location of the growth are illustrated in the X-ray and drawing (Figs. 2 and 3). Consultation was held with the Neurosurgical Department and the transcranial approach to this tumor was agreed upon as being the most favorable for the reasons already mentioned.

The patient entered the hospital December 11 and was prepared for operation the following day. The anterior half of the head and the eyebrows were shaved and prepared as for intracranial operations. The combination of local, avertin, and ether anesthesia was given. A long coronal incision was made within the hairline from the left temporal region over the vault and down into the right temporal region, as illustrated, and the scalp was peeled forward over the forehead down to the supra-orbital ridges. A frontal bone flap was then outlined on the right side (Fig. 6) similar to those used for pituitary tumor exploration. This flap was turned down without difficulty in the usual way. The temporal fascia and muscle formed a good pedicle. The dura was intact and slightly tense. A nick was made in the upper portion and a ventricle needle introduced to relieve tension.

The dura was now retracted from the roof of the orbit and the orbit was found to bulge upward more than in the usual case. In the course of turning down the flap, the upper portion of the frontal sinus had been opened and the osteoma could be seen to be present as illustrated. Beginning at the upper portion of the sinus, its posterior wall was removed as far as possible with the rongeurs, so that a portion of the medial part of the osteoma was exposed. It was impossible to carry this very far, however, because of the fact that the tumor was attached to the posterior wall of the sinus and, therefore, intimately adherent in this region. The motor burr was then used to drill into the roof of the orbit (Fig. 6d), removing bone to which the osteoma was attached. After this had been accomplished, it was possible to tilt the growth backward as it was not adherent elsewhere. The osteoma was grasped and lifted upward, brushing off the orbital tissue from its irregularities.

The nick in the dura was closed. The overhanging edges of the posterior wall of the frontal sinus were removed as well as its lining membrane in the upper and outer portion. This area of the sinus was obliterated by allowing the dura and brain to come forward. The mucous membrane in the medial portion and in the region of the nasal frontal duct was not disturbed and a probe could be readily passed into the nose. The roof of one ethmoidal cell had been removed. Its membrane was not disturbed.

All bleeding points were controlled. The bone flap was put back into place and the periosteum was sutured around the bone flap to hold it firmly. The scalp was then turned upward into place and the closure made in layers, leaving a small rubber tissue drain at each end of the incision.

Immediately following operation the head of the bed was somewhat elevated and the patient was cautioned against blowing the nose to prevent forcing air into the region formerly occupied by the frontal sinus. As a further precaution she was given ⅙ of morphia hypodermically. Otherwise, the pain and headache were relieved by aspirin and pyramidon. Luminal was sufficient to give rest for the night. The eyes were cleansed with 2 per cent boric acid solution. No other care was necessary except the dressings to the wound. Her postoperative course was quite uneventful. The temperature reached 100 on one occasion. Otherwise, it fluctuated between 98 and 99. There was a great deal of edema of the scalp and face which rapidly disappeared and was practically normal by the sixth day. The drains were removed on the second postoperative day and all sutures were out by the third day. There was considerable drainage of serum through the ends of the wound. The patient was allowed up on the sixth postoperative day and was discharged from the hospital 10 days after operation.

The patient returned to her work in 1 month and Figure 8 shows the result 50 days after operation. She also reported for check up on October 16, 1934, 10 months since the time of her operation. She was quite well in every respect and the right eye appeared to be back in the normal position.

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A USEFUL DIAGNOSTIC SIGN IN VERTEBRAL INJURIES[1]

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THE literature in recent years reviews a strikingly large number of vertebral injuries which have not been recognized until a very late stage when the condition has become irreparable. In a series of 90 cases of fracture of the spine, Stewart reported that 31 per cent of the fractures were overlooked. Fumagalli and Christopher found similar large percentages. These facts are unfortunate because, with the method of treatment by hyperextension, the results obtained in recent cases are fairly satisfactory. Early diagnosis, therefore, is obviously a necessity for the elimination of deformity and the reduction of permanent disability.

The diagnosis of vertebral fracture is frequently overlooked for several reasons. At the time of injury the clinical picture is clouded by damage to muscles and ligaments, which gives diffuse pain and extensive muscle spasm. The symptoms of fracture, on the other hand, may be extremely mild in character, and the patient is treated conservatively until, with disability persisting, a roentgenogram is taken which reveals the true pathological condition. The mechanism of the trauma may itself be of such slight degree that suspicion of the vertebral damage is not aroused. This is well illustrated by one of our cases in which the mere mechanism of stooping to pick up a light piece of wood produced a compression fracture of the twelfth dorsal vertebra without evident pre-existing pathological change. Another patient in our series, aged 38, fractured the third lumbar vertebra doing her habitual, but very active, morning exercises.

Often in patients with multiple fractures, such as are commonly seen in modern automobile accidents, the picture of shock and the problems of reduction of fractures in the extremities distract the surgeon's and the patient's attention so that the spinal injury is not recognized until the patient is ambulatory. In our series there are several patients, who were seen by us long after injury, who belong in that group. Diagnosis is usually made clinically by pain, limitation of spinal motion and tenderness; symptoms and signs which are generalized and which may be the result of other lesions than a vertebral injury. Frequently, in order to execute the routine examination of the back, the patient must be moved about and handled in a manner which is not only painful but sometimes harmful.

For several years we have employed a maneuver which is extremely helpful in the diagnosis of vertebral injuries. Although it is exceedingly simple, results can be obtained only if every detail of its technique is observed. The patient is placed flat on his back without pillows, the examiner places one hand upon the sternum of the patient exerting a slight pressure so that no flexion can take place either at the lumbar or dorsal regions of the spine; at the same time the examiner's other hand is placed under the occiput and with this hand the head is bent upon the neck, then slowly but forcibly, the head and neck are flexed upon the sternum (Fig. 1). This produces a progressive pull upon the posterior spinous ligaments, starting at the ligamentum nuchæ and being transmitted downward to the interspinous ligaments until it reaches the spinous process of the injured vertebra. On this it acts as a lever gently compressing the body and producing pain which the patient localizes very accurately (Fig. 2). The rôle of the interspinous ligaments is illustrated by Case 2 of our series. This man had a fracture dislocation of the first lumbar vertebra and presented a positive neck flexion test well localized at this area. A laminectomy was performed to relieve pressure on the cord, and following this procedure this test became negative. Watkins, in a paper on low back pain published in 1931, described briefly but accurately, this maneuver as the "Soto-Hall sign." Following a personal communication from us he had employed this test and mentioned its usefulness. Munzer, in 1933, described a head-bending test which he found valuable in inflammatory, trau-

Fig. 1. The maneuver used to detect vertebral injuries.

[1]The experimental part of this paper was conducted under a grant from the Christine Breon Fund for Medical Research of the University of California.

TABLE I.—RECENT FRACTURES OF SPINE—UNDER ONE MONTH

Case No.	Sex	Age	Period since injury	Signs and symptoms	Clinical diagnosis	Neck flexion test
1	F	24	1 day	Tenderness over lumbar spine and pubis	Fracture of spine L 1	Pain over L 1
2	M	43	1 day	Anesthesia of legs	Fracture of spine with cord symptoms	Pain over L 1; test became negative at once following laminectomy
3	F	57	1 day	Kyphos, low back pain	Fracture of spine L 1	Pain over L 1
4	F	25	2 days	Tenderness over L 5	Fracture of spine L 5	Pain over L 5
5	F	34	1 day	Severe low back pain	Fracture of spine L 2	Pain over L 3; still positive four years later
6	M	23	1 day	Tenderness over lumbar spine	Fracture of spine D 12	Pain over L 3
7	F	33	1 day	Tenderness over D 10	Fracture of spine D 10	Pain over D 8
8	M	45	2 days	Tenderness over dorsolumbar	Fracture of spine D 11 and 12	Pain over D 11
9	M	47	1 day	Tenderness over D 4 and 5	Fracture of spine D 5 and 8	Pain over D 5 and 8
10	M	28	3 days	Tenderness over L 1 and 3	Fracture of spine L 1	Pain over L 1
11	M	29	1 day	Tenderness over mid-dorsal	Fracture of spine D 8	Pain over D 8
12	F	50	1 day	Tenderness over D 5 and 7	Fracture of spine D 5	Pain over D 6
13	M	30	1 day	Tenderness over lower lumbar	Fracture of spine L 4	Pain over L 4
14	M	31	3 days	Tenderness over mid-lumbar	Fracture of spine L 2	Pain over L 2
15	M	23	1 week	Tenderness over lower dorsal	Fracture of spine D 9	Pain over D 9
16	M	50	2 days	Tenderness over mid-lumbar	Fracture-dislocation of spine L 3	Pain over L 2 and 3
17	M	19	3 weeks	Tenderness over mid-dorsal	Fracture of spine D 6	Pain over D 7
18	M	52	3 weeks	Tenderness over lumbodorsal	Fracture of spine D 11	Pain over D 11
19	F	50	1 month	Pain over right scapula	Fracture of spine D 12	Pain over D 8
20	F		1 month	Lumbar back pain	Fracture of spine L 1 and 3	Pain over L 2

Total number of vertebral fractures 20
Percentage, positive neck flexion test..... 100

matic, and various neurological disturbances, and believes that this head bending test is positive when the roots are involved as in infectious myeloradiculitis.

We have employed this sign in a large number of cases during the past 4 years and have tabulated a series of 58 consecutive cases in order to determine its value in differential diagnosis. To facilitate this evaluation we have arranged these cases in the form of tables showing the relationship of the neck flexion test to the clinical and radiological findings (Tables I to V). The sign was found to be uniformly present (100 per cent) in patients with recent compression fractures of a vertebral body which could be demonstrated by the X-ray. It has been our experience that a negative test within 1 month of an injury to the back was conclusive evidence of the absence of vertebral fracture. A well localized positive test is strongly suggestive of such an injury, but may be found in other acute conditions. In carrying out this maneuver one should not interpret as a positive test the discomfort and tautness near the cervicodorsal junction of which some patients complain.

In cases of acute sprain of the back, we have found a small percentage, consisting of the more severe ones, in which the sign is positive. In these it was noted that the pathological changes were mainly in the lumbosacral area, whereas most sacro-iliac sprains gave a negative sign. This sign is also positive in vertebral tuberculosis during the active stage and in tumors when some damage has occurred to the body of the vertebra. We have not followed a sufficient number of these cases of tuberculosis or tumor to determine its value in diagnosis, or its worth in estimating healing or the prognosis, which should be studied.

When injuries to the back are so commonly found associated with medico-legal problems, it is important to attempt to elicit information by methods which do not involve directly the suspected region. In such cases this maneuver has great value since information can be obtained without attracting attention by local examination of the injured area.

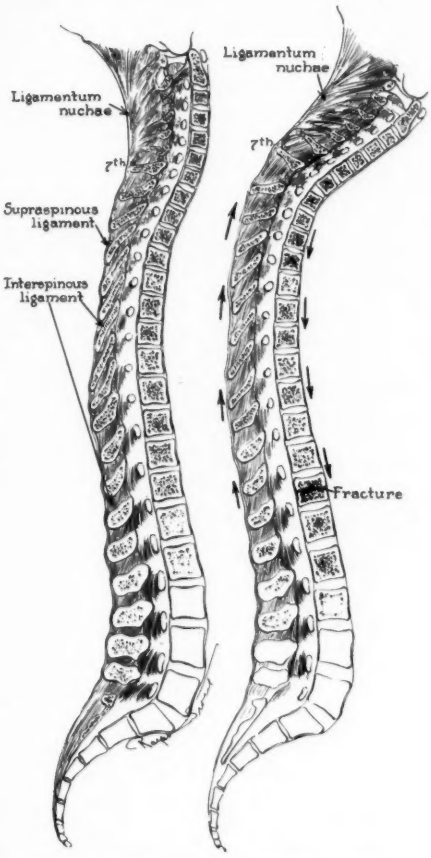

Fig. 2. A drawing explaining the maneuver.

EXPERIMENTS ON THE TRANSMISSION OF IMPULSE FROM THE CERVICAL SPINE TO THE DORSO-LUMBAR JUNCTION

To obtain experimental confirmation of the previously described clinical observations, a series of experiments was carried out on three rabbits. These experiments were performed along similar lines and with identical results, so that only one of the series need be described. A large male rabbit was killed with ether, the skin and superficial fascia were removed from the back and fine needles were inserted into each of the spinous processes from the third dorsal to and including the first lumbar. The animal then was firmly attached to a wooden board by means of a nail driven through its pelvis and other nails placed just anterior to the vertebral bodies in the cervicodorsal junction and in the lumbar region. Further immobilization of the dorsolumbar region was accomplished by the firm pressure of a piece of wood extending along the dorsal surface of the animal. Without changing the position of the thoracic or lumbar region, the following procedures were carried out and roentgenograms were taken.

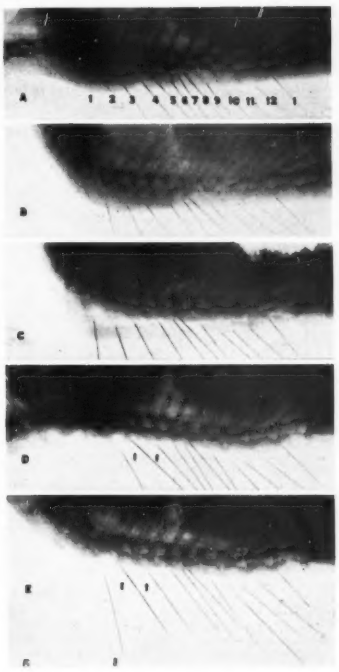

Fig. 3. A, Neck extended. B, Neck flexed. C, Neck flexed, back muscles removed. D, Neck extended, back muscles removed, ligaments cut between T_3-T_4 and T_4-T_5. E, Neck flexed. Same as D.

Procedure I. The neck was extended (Fig. 3 A). The neck was then flexed (Fig. 3 B). Upon carrying out the latter maneuver the spinous processes in the dorsal region showed a progressive separation in the manner of the opening of a fan, which movement was demonstrated more clearly by the excursion of the needles which had been inserted in the spinous processes.

Procedure II. The muscles of the back were removed on both sides of the spine. The neck then was flexed as shown in (Fig. 3 C). Movement occurred in the spinous processes and the corresponding needles in the same manner as in Procedure I.

TABLE II.—SPRAINS OF BACK

Case No.	Sex	Age	Duration of symptoms	Signs and symptoms	Clinical diagnosis	Neck flexion test
21	M	47	1 day	Rt. sacro-iliac tenderness	Rt. sacro-iliac sprain	Not painful
22	M	40	1 day	Lt. sacro-iliac tenderness	Lt. sacro-iliac sprain	Not painful
23	F	20	1 day	Lt. sacro-iliac tenderness	Lt. sacro-iliac sprain	Pain over lt. sacro-iliac
24	M	32	1 day	Rt. lumbar tenderness	Rt. lumbar muscle sprain	Not painful
25	M	39	1 day	Lumbodorsal pain	Sprain of erector spinæ muscle	Not painful
26	M	22	1 day	Rt. sacro-iliac tenderness	Rt. sacro-iliac sprain	Not painful
27	M	29	2 days	Lumbosacral pain on extension	Lumbosacral pain	Not painful
28	M	39	2 days	Lt. sacro-iliac tenderness	Lt. sacro-iliac sprain	Not painful
29	M	58	4 days	Rt. lumbar pain	Rt. lumbar muscle sprain	Not painful
30	F	32	1 week	Tender over 4th lumbar	Iliolumbar sprain	Pain over 4th lumbar
31	M	34	2 weeks	Tender over lumbosacral	Lumbosacral sprain	Not painful
32	M	58	2 weeks	Tender over rt. sacro-iliac	Rt. sacro-iliac sprain	Not painful
33	F	51	2 weeks	Lumbar pain and tenderness	Lumbar muscle sprain	Not painful
34	M	34	1 month	Tender over rt. sacro-iliac	Rt. sacro-iliac sprain	Pain over rt. sacro-iliac
35	M		1 month	Tender over D 7, 8, and 9	Mid-dorsal sprain	Not painful
36	M	44	6 weeks	Tender over sacrum and sacro-iliacs	Sacro-iliac sprain and sacral myofascitis	Pain over sacrum
37	M	25	6 weeks	Right sciatic pain	Rt. sacro-iliac sprain	Not painful
38	M	45	6 weeks	Lumbosacral pain and tenderness	Lumbosacral sprain	Pain over lumbosacral
39	M		2 months	Lumbosacral pain and tenderness	Lumbosacral sprain	Pain over lumbosacral
40	F	22	10 weeks	Tender over lt. sacro-iliac	Lt. sacro-iliac sprain	Not painful
41	M	56	3 months	Tender over rt. sacro-iliac	Rt. sacro-iliac sprain	Not painful
42	M	18	5 months	Lt. sciatic and sacro-iliac pain	Lt. sacro-iliac sprain	Pain over lt. sacro-iliac

Total number of cases..... 22
Percentage, positive neck flexion test..... 32

TABLE III.—ARTHRITIS OF THE SPINE

Case No.	Sex	Age	Duration of symptoms	Signs and symptoms	Clinical diagnosis	Neck flexion test
43	F	42	1 month	Tender over C 7 and upper dorsal	Arthritis of spine, upper dorsal	Pain over C 7
44	M		2 months	Lumbosacral pain	Arthritis of spine, L 4 and 5	Pain over lumbosacral
45	M	60	7 months	Rigid spine, lumbodorsal	Arthritis of spine, lumbodorsal	Not painful
46	M	50	8 months	Tender over D 8, 9, 10	Arthritis of spine, dorsal	Not painful
47	F	48	Years	Backache, tender over D7 and 8	Arthritis of spine, mid-dorsal	Not painful

Total cases..... 5
Percentage, positive neck flexion test..... 40

Procedure III. All the posterior ligaments between the third and fourth dorsal vertebræ were cut down to the cord; also between fourth and fifth, as shown by arrows in Figure 3, D and E.

Following the severance of these ligaments it was seen that flexion of the neck produced a separation of the spinous processes only as far distally as the fifth dorsal vertebra, beyond which no movement occurred (Fig. 3 E).

Procedure IV. A laminectomy was done at the level of the third and fourth dorsal vertebræ. Flexion of the neck then produced a slight movement of the fifth dorsal vertebra, which movement no longer occurred after the bodies of the second and third dorsal vertebræ were removed. The spinal cord shifted slightly cephalad on flexion of the neck.

EVALUATION

The foregoing procedures indicate that the movement of flexion of the neck causes a tension

TABLE IV.—TUBERCULOSIS OF THE SPINE

Case No.	Sex	Age	Duration of symptoms	Signs and symptoms	Clinical diagnosis	Neck flexion test
48	M	28	3 months	Pain and kyphos at D 7	Tuberculosis of spine, D 7 and 8	Pain over D 7. Test became negative 15 months after Hibb's fusion
49	M	54	6 months	Pain in back, paraplegia	Tuberculosis of spine, D 10 and 11	Pain over L 1
50	F	19	1 year	Scoliosis, tender rt. sacro-iliac	Tuberculosis of spine, D 12	Pain over L 2
51	F	24	1 year	Tender over L 3 and 4	Tuberculosis of spine, L 3 and 4	Pain over lower lumbar. Test became negative 1 year after Albee fusion

Total cases..... 4
Percentage, positive neck flexion test..... 100

TABLE V.—MISCELLANEOUS

Case No.	Sex	Age	Duration of symptoms	Signs and symptoms	Clinical diagnosis	Neck flexion test
52	M	28	1 day	Tender over lt. costovertebral angle	Fractured arthritic spur on D12	Pain over L. border of L 1
53	M	28	5 days	Pain over mid-lumbar	Fractured arthritic spur on L 3	Pain over L 3
54	M	60	6 months	Kyphos and tender over D11 and 12	Metastatic carcinoma of spine D12 and L 1	Pain over D 11 and 12
55	F	26	3 months	Low dorsal pain and tenderness	Contusion of back	Not painful
56	F	68	3 months	Tender interscapular swelling	Contusion of back	Not painful
57	M	24	1 week	Mid-dorsal tenderness and kyphos	Osteochondritis of spine	Pain over mid-dorsal
58	M	39	5 months	Tender over D 4	Kuemmel's posttraumatic syndrome	Pain over D 4 dorsal

Total cases..... 7
Percentage, positive neck flexion test..... 71

upon the ligamentum nuchæ, the supraspinatus ligament, and the interspinous ligaments by means of which a progressive slight separation of the spinous processes of the dorsal region accompanies flexion of the neck. This takes place in the absence of the muscles of the back. The amount of movement, however, is greatly limited after the excision of all ligaments. An almost negligible motion of the vertebræ occurs as a result of the transmission of the impulse through the vertebral bodies.

To summarize, these experiments give an explanation of the fact which has been observed clinically and reported in the accompanying tables namely: that flexion of the neck, mainly by the transmission of ligamentous pull, causes pain which is localized at the level of the injured vertebral body when it is the site of a compression fracture or destructive process.

SUMMARY

The usefulness of this sign rests upon the following facts:

1. It gives the exact location of recent fractures without moving or disturbing the patient.
2. It guides the technician in obtaining roentgenograms at the proper level.
3. It aids in the differentiation between the injury of bone and damage to soft tissue.
4. It is helpful in medico-legal cases because it does not attract the attention of the patient to the lesion and he is not aware of the area that is being tested.
5. A negative test almost eliminates the diagnosis of recent vertebral injury.
6. Confirmation of the clinical observations is given by experiments on rabbits.

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A NEW OPERATIVE PROCEDURE FOR BRACHIAL BIRTH PALSY—ERB'S PARALYSIS

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SEVERAL operations have been used for correcting the internal rotation deformity of Erb's palsy and so improving the position and to a certain extent the function of the shoulder. Among these are the section of the subscapularis tendon devised by Sever, the rotation osteotomy of the humerus devised by Hibbs, and the subperiosteal rotation of the humerus by Kleinberg. All of these correct the deformity, but in a sense are negative operations; that is, they do not attempt to restore the action of the deltoid except indirectly. It was in an attempt to do this that the operation here described was devised.

The majority of cases of brachial birth palsy are of the upper arm type and in these there is usually considerable power left in the deltoid. It was noted that in these cases the anterior portion of the deltoid is frequently almost normal in power while the posterior portion is practically powerless. The operation to be described is an attempt to utilize a portion of this strong anterior deltoid to reproduce more nearly the normal deltoid action, by restoring the balance between the anterior and posterior portions. To accomplish this a portion of the origin of the anterior deltoid is shifted backward to replace the paralyzed posterior portion and by thus changing its direction of pull, restore the normal deltoid action, at least in part.

Technique: The patient lies on the operating table with the shoulder to be operated on elevated by a sand bag placed well under the scapula. The incision commonly used is transverse, extending from about the middle of the spine of the scapula to a little beyond the anterior end of the acromion process. This incision reaches the ridge of the scapular spine behind and the lateral portion of the anterior half of the deltoid. At times the anterior end of the incision is extended downward along the muscle for an inch or two. The superficial fascia is stripped back from the muscle for a distance of 2 or 3 inches, but the muscular sheath is not disturbed. The next step is preparation of a bed in the spine of the scapula for the reception of the transplanted portion of the deltoid. This is done by splitting the periosteum over the ridge of the scapular spine at about the place where it begins to spread out into the acromion process medially for about 1½ inches. The periosteum is then stripped from both sides of the scapular spine and a portion of the bone of the spine is gouged out. Next the portion of muscle to be transplanted is prepared. Care must be taken to take good healthy functioning muscle. We have been able to secure sufficient by cutting off the outer border of the acromion process with the attached muscle. The process being cartilaginous in children is easily divided with a knife or sharp osteotome. After separating the tip of the acromion the deltoid muscle is split downward along the course of the fibers by blunt dissection. Care must be taken not to divide too far downward in order not to interfere with the nerve supply of the remaining anterior portion of the deltoid. It seems to be safe to divide it approximately half way from the acromion process to its insertion in the humerus. At this stage of the operation, the floor of the incision is formed by the capsule of the joint and, if the subscapularis muscle is tight, it can be divided, as in Sever's operation. This has been necessary in some cases. After the muscle strip has been prepared, the arm is abducted laterally and externally rotated to allow the separated acromion, with the strip of muscle attached, to be fitted into the bed prepared for it in the scapular spine. It is then attached by chromic catgut sutures to the spine of the scapula. The periosteum stripped from the spine is also sutured to the edges of the separated portion of the acromion with chromic catgut. The arm must be held in the abducted and externally rotated position from this point on through the application of the cast. Closure of subcutaneous tissue is done with catgut and the skin with silkworm. A cast over the body and arm is then applied with the arm in the position mentioned.

The cast is left on 6 weeks, after which the portion over the shoulder and arm is bivalved and the upper portion removed, the rest being left as a splint. The patient then exercises the arm by moving the elbow and raising it from the splint. After 2 weeks the body portion of the cast is also bivalved so that it can be removed for increased motion of the shoulder, and replaced. After 2 weeks it is completely removed. At this time the humerus is held fixed in abduction on the scapula, somewhat as in a shoulder fusion. This we have

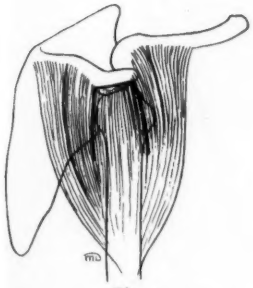

Fig. 1.

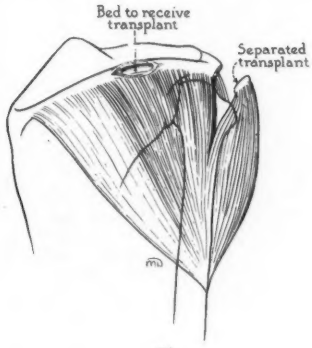

Fig. 2.

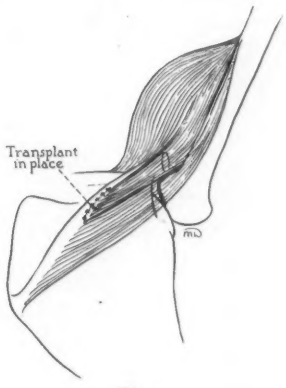

Fig. 3.

Fig. 1. Diagram showing portion of deltoid to be transplanted.

Fig. 2. Diagram showing transplant separated and bed prepared in scapular spine.

Fig. 3. Diagram showing transplanted muscle sutured to scapular spine. Humerus abducted and externally rotated.

attributed to the fact that the transplant is somewhat short for its new position. We have felt that it was better to let the necessary adaptive lengthening take place gradually rather than to attempt to hasten the process by stretching. The length of time for this varies but usually takes 2 or 3 months. The transplanted muscle seems to act as an external rotator largely, and this gives the principal improvement in function of the arm.

The operation, of course, is of no value in cases with complete deltoid paralysis. Its field is limited to those cases in which the anterior deltoid is strong, and in these it has been effective in restoring the muscle balance to a certain extent.

We have had one flat failure. This we ascribed to the fact that the child was too young at the time the operation was performed. The tissues were very soft and it is possible that the transplant did not hold. Also he was too young to co-operate successfully in exercises.

The operation has been done on 7 patients, all at the Shriner's Hospital.

Case 1. R. H. aged 13 years, was operated upon January 6, 1930. The patient had the usual internal rotation deformity of the shoulder. In this case there was a definite backward subluxation of the humeral head from the glenoid cavity. Passive motions of the shoulder joint were quite free, i.e., there were no contractions of the subscapularis or pectoralis. There was some flexion contracture of the elbow joint from the biceps contracture. The humerus was somewhat shortened. Active abduction of the shoulder laterally was nil, anteriorly to a right angle. In this case the subluxation of the humeral head attracted attention and in trying to think out a means of preventing it, transplantation of a portion of the deltoid backward suggested itself, with the idea of reinforcing the posterior portion of the joint capsule. On removal of the cast the improvement in function was so noticeable that it seemed wise to try the procedure on other cases. In a way the improvement in the shoulder function was a by-product. This patient was last seen April 24, 1934. He can now abduct the arm laterally to a right angle and externally rotate it about 45 degrees. To his mind the greatest improvement

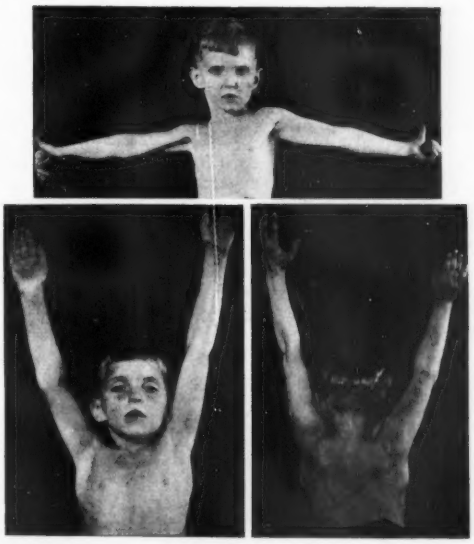

Fig. 4. Case 3. C. H. Good result. a, Before operation, showing maximum abduction and external rotation of right arm. b, After operation, showing abduction. c, Back view, showing abduction. Note the shortening of the upper arm and forearm.

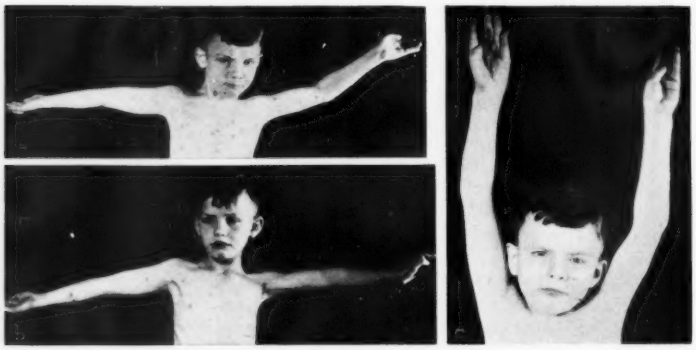

Fig. 5. Case 4. R. P. Good result. a, After operation, showing improved external rotation of shoulder. b, Before operation, showing maximum abduction and external rotation of left shoulder. c, After operation, showing improved abduction of shoulder.

is that he can now throw his hand up enough to catch a baseball.

CASE 2. D. B. age 3 years. January 7, 1932. The patient presented the usual internal rotation deformity at the shoulder. Passive motions of the joint were free. Active lateral abduction was about 20 degrees. Anterior abduction was possible to a right angle. External rotation was nil. This case was a failure, we believe, because patient was operated upon at too early an age. The tissues were very soft and it is possible the transplant may not have held.

CASE 3. C. H., aged 5 years, was operated upon January 26, 1932. There was the usual internal rotation deformity of the shoulder with moderate shortening of the humerus. Passive motions were free. Active abduction laterally was possible to slightly less than a right angle; to right angle anteriorly. External rotation was nil. Patient was last seen January 12, 1934, at which time he could raise the arm above his head nearly as far as the normal one. External rotation was 90 degrees.

CASE 4. R. P., aged 7 years, was operated upon August 2, 1932. The usual internal rotation deformity, with moderate shortening of humerus, was noted. Passive motions were free. Active motion lateral abduction to right angle—anterior abduction to right angle. External rotation was nil. Patient was last seen June 16, 1933, and could now raise arm practically straight over head. External rotation was 90 degrees.

CASE 5. C. P., aged 11 years, was operated upon April 6, 1933, for the usual internal rotation deformity. Passive motion was only slightly limited (had had Sever operation previously). Active motions—lateral abduction was about 15 degrees, anterior abduction about 80 degrees or less. External rotation was nil. In this case the lower arm was also affected. Supination of the forearm was lost and flexion of the first two fingers was weak. Patient was last seen June 22, 1934, when he could place the hand back of head. Lateral abduction was possible to a right angle; external rotation to about 60 degrees. The transplant could be felt functioning.

CASE 6. J. R., aged 7 years, was operated upon July 25, 1933. He had the usual internal rotation deformity. Passive motions were considerably limited. Abduction was possible to somewhat less than a right angle laterally. External rotation was markedly limited. Active motions—

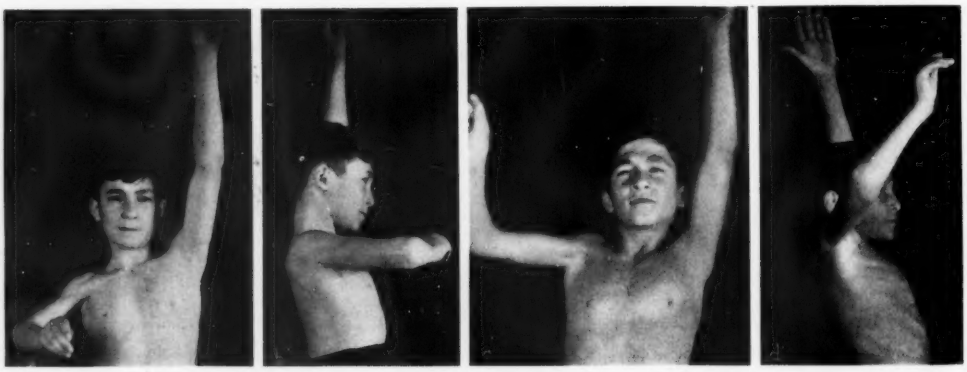

Fig. 6. Case 5. C. P. Fair result. a, Before operation, showing maximum lateral abduction and external rotation. b, Before operation, showing maximum active anterior abduction. c, After operation, showing improved active abduction and external rotation. d, After operation, showing improved active external rotation.

lateral abduction was about 30 degrees; anterior abduction, about 80 degrees; external rotation was nil. On account of the marked limitation of passive movement, a Sever operation was done in connection with the deltoid transplant in this case. Patient was last seen April 25, 1934, when lateral abduction was possible to a right angle. He could rotate shoulder externally enough to place hand on head with shoulder abducted laterally. This patient was one in whom the motion of the shoulder joint was somewhat slow in returning. He has developed a somewhat peculiar technique of using the arm by fixing the head of the humerus with the transplant and then moving the scapula.

CASE 7. J. M., Aged 6 years, was operated upon October 12, 1933. He had the usual internal rotation deformity. Passive motions were poor. (Had had Sever operation 1 year previously.) Active motions—lateral abduction about 20 degrees; anterior abduction, nearly 90 degrees; external rotation, nil. Patient was last seen July 26, 1934. He could then abduct the arm laterally to right angle and could rotate externally enough to place the hand on top of the head. Motion of the shoulder joint was returning rather slowly.

ANALYSIS OF CASES

Of the 7 cases, 3 can be classed as much improved, 3 as considerably improved, and 1 as a failure. The more favorable cases for the operation seem to be those in which there is a fair amount of lateral abduction present. In these cases there is at least a fair amount of power present in the portion of the deltoid originating from the acromion. Hence, when this portion is transplanted backward it functions more vigorously. The most noticeable improvement has been in the external rotation. In 2 cases a splint of the usual obstetrical paralysis type was applied for some time after the removal of the cast. In these 2 cases, stiffness of the shoulder joint was slower in disappearing.

Fig. 7. Case 6. J. R. Fair result. a, Before operation, showing maximum active abduction and external rotation of right shoulder. b, After operation, showing improved abduction and external rotation. c, After operation, hand placed actively behind head.

While the series of cases reported is small and too recent to state definitely what the long range result will be, yet the procedure has shown improvement in the function of the arm.

POSTOPERATIVE OR VENTRAL HERNIA

A Method for Relief of Tension after Repair

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THE procedure about to be described is contributed to the already voluminous literature on postoperative or ventral hernia, with the firm conviction that it will decrease the number of failures of this often formidable operation and will bring some apparently inoperable cases within the field of operability. It can also be used in cases in which, because of obesity and high intra-abdominal tension, postoperative hernia is likely to develop. The device may perhaps be crude and amenable to refinements of application, but it is believed that the principle is sound and the method has proved effective in actual practice.

The majority of abdominal incisions are made in the longitudinal axis of the body. They are popular because of the increased ease of exposure over the transverse incision in spite of the fact that this transverse incision avoids nerves and is parallel to the direction of the fibers of the transversalis fascia on which the integrity of the abdominal wall depends. Intra-abdominal tension is horizontal or at right angles to the longitudinal axis of the body. Chiene aptly described it as a tug of war going on between the two groups of oblique muscles of the abdominal wall. It is this constant drag away from the midline of the abdominal wall that puts tension on suture lines used to repair a longitudinal incision, thus causing ischemia of wound edges and necrosis. It also causes sutures to cut through, produces serum in wounds leading to infection, and makes healing difficult and scars defective.

It is much more important to direct effort to relieving wound tension than it is to devise some new type of suture material. A wound that is completely relaxed and without tension will have a good blood supply and no matter what suture material is used, whether absorbable or non-absorbable, or it might almost be said, whether any at all is used, the wound will heal. In practice a moderate amount of abdominal tension is not inimical to wound healing and especially if some tension sutures are so placed as to relieve the strain on the wound margins.

It is in the obese patient, however, with a high degree of intra-abdominal tension that we have our difficulties, and if the hernia is large and, as is more than likely, in the upper abdomen, we have a situation that often borders on the inoperable and in which there is a high surgical risk and numerous failures. Reduction in weight helps to lessen intra-abdominal tension but such efforts are often only partially successful.

THE DEVICE

The device here illustrated is a heavy canvas belt similar to that long used for fracture of the pelvis. It is wide enough to include the lower ribs and the hips. The ends come to slightly above the level of the abdominal wall and are split into tails so as to more closely follow the contour of the body and prevent all the tension being taken up by the hips and the ribs. A row of small pulleys is attached to either end of this belt and a small sash cord laced through these pulleys alternating with a row of pulleys attached to a metal bar a short distance above the abdominal wall (Fig. 1). The amount of squeezing together of the abdominal wall can be somewhat regulated by the distance of this upper row of pulleys from the abdominal wall. The bar is then lifted by a cord attached to either end and threaded through a pulley and the tension maintained by weights attached to a cord running through pulleys on the overhead frame of a fracture bed (Fig. 2). About 20 pounds of weight is all that has ever been found necessary. The head of the bed can be elevated to suit the patient's comfort. The patient can turn to either side without increasing the tension on the wound and, what is of great importance, the wound can be dressed without increasing the strain on the sutures as would be the case if adhesive strapping had to be cut and the abdominal wall allowed to sag to either side. More kindly healing is shown by the lessened amount of serum and infection in the wound.

The purpose of the sling is to lift the weight of the sides of the abdominal wall and also to compress the hernial margins toward the midline. Of the two the lift is the more important and does the most to relieve tension. The time-honored method of adhesive strapping fails to do this and permits of dragging each way from the midline. This method also fails to keep up a constant, even relaxation of the abdominal wall. The apparatus here described does accomplish this by following

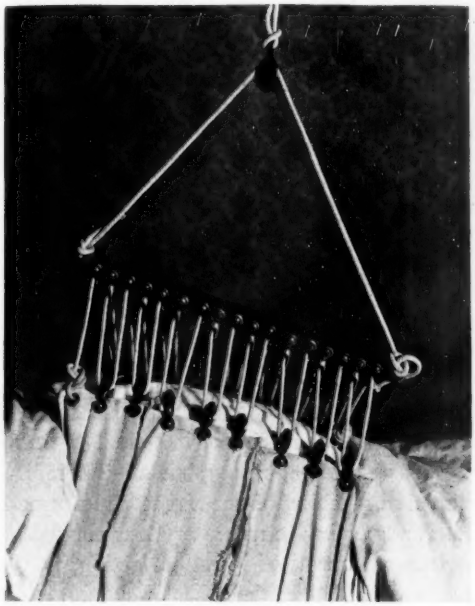

Fig. 1.

every move of the patient with an even lift as in fracture extension. The efficiency of this method may be demonstrated by strapping an obese abdomen with long strips of adhesive tightly enough to approximate the edges of the ventral hernia. If the patient is then placed in this sling the adhesive can be easily relaxed to a marked degree by a lift of 15 to 20 pounds. The adhesive thus becomes unnecessary.

The early discomfort of which some patients will complain when the apparatus is first put on can be greatly lessened by its application for a week in advance of operation. During this time the patient becomes accustomed to bed regimen and gets over a great deal of the restlessness which is usually present after being first put to bed. The very real danger to life which accompanies repair of large ventral hernias comes largely from the increase in intra-abdominal pressure. This produces pressure on the diaphragm and embarrassment of respiration making hypostatic pneumonia a not uncommon complication. Alarming

Fig. 2.

cardiac symptoms from pressure on the heart and abdominal vessels are often seen. Acute dilatation of the stomach and ileus may complicate the picture and further increase the pressure. This may all be largely avoided by this period of preoperative preparation. The abdomen should be strapped with wide adhesive tightly enough to approximate the edges of the hernial ring and reduce its contents into the abdomen. The sling is then applied and enough weights added to lift the abdominal wall and obtain considerable relaxation of the adhesive strapping. Several days should then intervene before operation. The patient then becomes accustomed to the increased intra-abdominal pressure. There should be no embarrassment of heart action or of respiration, no vomiting, and the bowels should be functioning normally, before operation should be considered. If untoward symptoms arise and the patient cannot tolerate this program, it should be a warning that surgical repair is risky or impossible and should thus prove a valuable test of operability.

When this principle was first being tried out this apparatus was applied after the patient had returned from the operating room. Often this increased the postoperative discomfort and pressure against the diaphragm and heart to such a degree that it had to be removed. Since this period ot pre-operative preparation has been carried out, no patient has developed alarming postoperative symptoms, for he has become accustomed to the apparatus beforehand, and the postoperative course has been smooth.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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THE MESENTERY

TEXTBOOKS of anatomy devote but little space to one of its most extraordinary and important parts, the mesentery. Casual inspection might give the idea that the mesentery is merely a ligament to maintain the small intestine loosely in its variable position. It is true that the mesentery performs this function, but in so doing an anatomical structure is presented difficult to imitate in human made apparatus. The root of the mesentery, or its parietal attachment, where apparently it arises from the posterior abdominal wall, is only 15 centimeters long, whereas its visceral attachment, where its layers divide to enclose the full length of the jejuno-ileum, is nearly 7 meters long. In other words, the mesentery in extending an average of 20 centimeters from its origin to its intestinal attachment, increases in length forty-six times, a result attained by marvellous "tucking." Through such a widespread distribution the superior mesenteric artery entering the mesentery at its root probably subdivides into more branches than any other artery in the body. Monks' description of the arrangement of the vascular loops in the mesentery is helpful in identifying different portions of the intestine.

Consider then the vital products of digestion which flow through the mesenteric veins and lymphatics—the total output of the chemical laboratory of the alimentary canal. In these vessels, inanimate foodstuffs first take on living properties. Just what biochemical changes occur as aminoacids, sugars, and fats pass through the intestinal wall do not yet seem to be fully explained. The interminable branching of the vagus and sympathetic nerves in the mesentery, exerting such significant control of intestinal action, is no less wonderful than the vascular supply.

From a surgical standpoint the mesentery is equally interesting and important. Tuberculous involvement of its glands (tabes mesenterica) may simulate tumors or appendicitis, or other abdominal diseases. Lymphosarcoma may be the cause of intestinal obstruction, while obstruction also may be due to volvulus or intussusception produced by defective mesenteric attachments. Again, calcified glands in the mesentery may be mistaken for calculi in the kidney or ureter until their mobility under the fluoroscope clears the diagnosis.

The absence of collateral circulation of mesenteric arteries must be constantly borne in mind in operations upon the mesentery and small intestine. If the excision of a cyst menaces the blood supply of a segment of bowel, the segment also should be removed. In intestinal excision and anastomosis for trauma or disease, disaster will follow failure to preserve vascular integrity. Especial care is necessary in an end-to-end anastomosis.

On account of the difficulty and urgency of recognition, no lesion of the mesentery excites more interest than thrombosis. Four vital organs are commonly responsible for fatal thrombosis and embolism—the brain, heart, lungs, and small intestine. No prophet can be bold enough to declare that surgery will never save a case of cerebral or coronary thrombosis. At present Trendelenburg's operation, or its modifications, occasionally may cure pulmonary embolism, but more often it is possible to preserve the life of a patient suffering from mesenteric thrombosis. Spontaneous recoveries have been reported, but the prognosis usually depends upon the time of operation and the amount of bowel involved. Excision of the damaged gut and primary closure is the operative method to be preferred, but often the exigency permits only enterostomy and exteriorization to be performed, sometimes with success.

While the mesentery does not rank as an organ, it is an essential part of the anatomy, and its position and functions and the pathological and traumatic processes to which it is so frequently exposed make it a structure of supreme concern in abdominal surgery.

FRANK K. BOLAND.

ATTACHMENT OF THE SKIN TO LESIONS OF THE BREAST

THE physical characteristics of attachment of the skin to underlying lesions of the breast are probably the most important clinical signs in distinguishing benign from malignant lesions. The skin is usually freely movable over benign tumors but usually is more or less fixed to underlying malignant growths. However, although this so called attachment of the skin to the tumor may be considered one of the most characteristic clinical signs of malignant disease, it is not pathognomonic, for a moderate degree of attachment may be present in some cases in which lesions are benign.

There are four degrees of fixation of the skin to the underlying growths depending on the duration of the lesion, its situation in the tissues of the breast, and the amount of intervening subcutaneous fat. In the first degree, the skin is only slightly attached to the tumor; on physical examination a slight pull on the skin, which does not move freely over the growth, causes dimpling at the point of attachment. In the second degree, the skin is fixed to the growth, and definite dimpling or retraction is seen in the skin over the tumor. This is often accentuated when the breast is elevated by raising the arm. In the third degree, the skin is firmly adherent to the underlying growth over a wide area, and the skin is often red as a result of beginning inflammatory necrosis. In the fourth degree, the skin is ulcerated and there is a discharge of necrotic material and pus as a result of secondary infection. The latter condition, secondary infection, is seen only in long standing cases; but unfortunately, such long standing cases are not uncommon inasmuch as 8 to 12 per cent of the cases encountered at the clinic each year are of this type.

Any degree of attachment of skin to a lesion is not an early sign of malignant disease. I recently reviewed more than 5000 cases of carcinoma of the mammary gland and found that, of the cases in which the skin was attached to the lesion, in 72 per cent there was metastasis to the regional lymph nodes. This indicates that if malignant disease is to be diagnosed early, the diagnosis must be made before attachment of the overlying skin is demonstrable. The only safe method by which to accomplish this is to remove the tumor for microscopic study immediately on its discovery.

In the presence of the third and fourth degrees of attachment, the clinical diagnosis of malignant disease is usually obvious. Fixation of the second degree is usually easily elicited by physical examination. Often it is difficult to detect attachment of the first degree, because the lesions are small and of relatively short duration and the degree of attachment, as has been said, depends on the situation of the growth in the tissues of the breast. If the tumor is situated on the posterior wall of the breast, there may be little or no attachment of the skin because the growth is attached to the underlying major pectoral muscle. If the lesion is situated on the anterior surface of the breast, attachment of the skin will be noted even if the lesion is very small. If there is a large amount of fat between the growth and skin, the fat may become lobulated, may give a soft feeling to the tumor, and in many instances may lead one to believe that the soft feeling area is larger than the tumor beneath. Tumors situated in the extreme margins of the breast often present unusual physical characteristics because of the difference in the character and distribution of the subcutaneous fat over the breast and over the adjacent thoracic wall.

The skin may appear to be attached to benign lesions of the breast because of the fold of skin in the region. Not uncommonly malignant growths along the extreme margin of the breast are not thought to be primary tumors but metastatic growths from lesions elsewhere. This is particularly true of growths situated in a region of accessory mammary tissue high in the axilla.

It is often difficult to detect the presence of cutaneous attachment to tumors that are situated close to the nipple or areola because of the contraction of the muscular fibers of the areola and nipple. These fibers often will make it appear that the skin is attached to the tumor when in reality such apparent attachment is a puckering of the skin brought about by contraction of the muscle. It is interesting to note the changes in the appearance in many of these cases at the time of operation, after the patient is anesthetized; the characteristic appearance of attachment cannot then be elicited because of relaxation of the muscular fibers of the breast.

Attachment of the skin to benign lesions of the breast is usually the same as the first degree attachment of malignant lesions. Cutaneous attachment to benign tumors usually is caused by an inflammatory reaction in the underlying mammary tissue, such as is seen in a region of purulent mastitis, fat necrosis, comedo-mastitis, or in a region of mastitis over a simple cyst. This last is probably the benign lesion that most commonly is diagnosed clinically as malignant disease. The error occurs not only because of the slight attachment of the skin to the underlying mammary tissue as a result of pressure, but also because of the hard, irregular feeling of the tumor as a result of the nodular mastitis that lies between the skin and the cyst. In many instances these cysts are found on the posterior wall of the breast, and when the breast is examined while the patient is leaning forward the rounded contour of the posterior wall of the cyst can be felt. In most instances these tumors are fairly large, and the absence of any enlargement of the regional lymph nodes suggests a benign lesion; a malignant tumor of as large size usually gives evidence of regional metastasis.

The most important malignant lesions of the breast, from a surgical standpoint, are early growths to which the skin is attached in slight degree. Inasmuch as this degree is also found in benign lesions, it is often difficult to make a definite clinical diagnosis on physical examination alone. As has been said,

the safest procedure to follow is to remove the growth for microscopic examination. The fact that approximately three of every four patients with malignant disease of the breast who present cutaneous attachment at the time of operation are found to have regional metastasis, indicates that such attachment is not an early sign of malignant disease of the breast, and that if improvement in surgical results is to be obtained, these growths must be removed before attachment is demonstrable clinically. STUART W. HARRINGTON.

LANDMARKS IN SURGERY

CARL LUDWIG SCHLEICH

His Contribution to Regional Anesthesia—An Historical Record

ALBERT H. FREIBERG, M.D., F.A.C.S., Cincinnati, Ohio

JUNE 8th was an eventful day for the surgeons of Germany, in the year 1892. On this day there was dedicated in Berlin the Langenbeck Haus, erected as the permanent home of the Deutsche Chirurgische Gesellschaft and as the future meeting place of its annual congress. At the invitation of a friend, I was privileged to attend some of the sessions during the dedication and to see in attendance many of those men of Germany whose names have meant so much in the development of surgery and its specialties. Among these might be mentioned Geheimer Ober Medizinal Rat, Professor Adolf von Bardeleben, chief of the I. Surgical Clinic of the Berlin Charité Hospital, the president of the congress; Thiersch, of Leipzig; Franz Koenig, then professor at Goettingen, later called to the chair in Berlin and author of that textbook on surgery which, in my opinion, is the best which any German has produced; von Bergmann; James Israel; Gurlt, who was secretary of the Congress; Trendelenburg; von Esmarch; Max Schede; Gussenbauer; Olshausen; Bardenheuer; Bier; von Eiselsberg; von Mikulicz, of Breslau; Julius Wolff, in whose clinic I was working; a younger man, later Wolff's successor—Albert Hoffa, the real founder of a modern group of German orthopedic surgeons, greatly beloved by all who knew him.

A paper which seemed to me of particular interest was that presented at a session on June 11, by Carl Ludwig Schleich, who was announced to speak on "Infiltration Anesthesia (Local Anesthesia) and its Relationship to General Narcosis." Schleich was a young man and rather handsome. According to his own memoirs[1], he was then 33 years old. His attitude was one of quite apparent assurance—perhaps too much so in view of the audience which he was addressing. (As a long time has elapsed since the occurrence of the episode which is being described, I have refreshed my memory by consulting the stenographic protocol which is found in the society's *Proceedings*.[2]) After an introductory section in which the analgesic effect of distilled water and various solutions was set forth, he described the use of very dilute solutions of cocaine in isotonic salt solution; dilutions varying from 1:5000 to 1:1000. The approach to the clinical portion of his paper was then made by the following, which is a fairly literal translation from the official protocol:

> Thanks to a perfected development of operative technique which we owe chiefly to our great masters of the pre-antiseptic period, not least to him whose immortal name this house bears, the dangers of an operation involving technical difficulties such as hemorrhage and traumatic damage may be said no longer to exist, provided there has been good training; Lister has made it possible for us to make the dangers of infection dependent upon our sense of responsibility and care; the dangers of intoxication by antiseptics to lay the necessary greater weight upon asepsis. What then, remains to be done most urgently? To reduce the dangers of narcosis!

With this statement a murmur went through the hall and it was not difficult to read cynicism in the men's faces. The paper concluded with a statement, however, the effect of which can not be entirely envisioned in its verbiage. For this one would require the personal impression which remains vividly with me to this day; an impression of great satisfaction with self, of the consciousness that something of perennial value was being handed over to posterity, as indeed it was in truth. Schleich's concluding sentence was:

> However, I must declare it to be entirely unjustifiable to perform operations under narcosis which might have been done under this, or a similar method of local, anesthesia; this not only from the moral and humanitarian standpoint, but with regard to the criminal responsibility of the surgeon.

The indignation which this final sentence aroused in the president, von Bardeleben, was at once apparent. His florid countenance became suffused with a deeper glow, amounting almost to cyanosis. With evident trembling, he rose to his feet. The auditors were murmuring to one another. The moment was one of great dramatic tension as the president found voice to say:

> In our society we are not accustomed to taking a vote in a matter of this kind. When, however, such a statement is hurled at us as is contained in the concluding sentence of him who has just spoken, we have a right to express our opinion, for this is a public meeting. I ask those who are convinced of the propriety and truth of the conclusions expressed by him in his last sentence, to raise their hands. Gentlemen, I observe that no hand has been raised. Is a discussion desired?

Immediately there was a roaring "nein" which seemed to come from the throats of all of the members. There ensued a period of utmost confusion

[1] Besonnte Vergangenheit. Berlin: Rowohlt Verlag, 1926.

[2] Verhandl. d. deutsch. Gesellsch. f. Chir., 1892, xxi congress.

in the assembly. It seemed as if everyone present were talking to someone else. The president rang, long and loudly, the large hand bell which stood on his table and which served in place of the chairman's gavel to which we are accustomed. Presently the uproar subsided sufficiently for the president to be heard. He said: "Will such as favor a discussion, raise their hands? No hand is raised and there will, therefore, be no discussion."[1]

Carl Ludwig Schleich
1859–1922

Schleich had advanced to ask for the floor and this was the occasion for the uproar. There was now nothing for him to do but to withdraw from the hall. As he approached the entrance, there arose from his seat a few rows in front of us a gentleman with a mane of gray hair, who followed Schleich through the door. I learned that this was his father, an ophthalmologist in Stettin, who believed in the statements of his son. It should be added that there was a rumor that the mortality record of the I. Surgical Division of the Charité Hospital from chloroform administration was far from good; it was a sensitive point. Even more, it was said that von Bardeleben had had a death from this cause shortly before the meeting.

One year later, at a meeting of the Surgical Society, Schleich was given the opportunity to demonstrate his method. Only 30 men responded by their presence. It was Professor Ernst von Bergmann who extended the invitation, fighter for the establishment of scientific truth that he always was. Aristocratic to his finger tips, von Bergmann was always to be counted on as a virile antagonist to prejudice, to littleness, to niggardly appreciation of merit when questions of scientific and professional advance were involved. And on this very occasion, he did not hesitate to oppose another aristocratic surgeon, von Esmarch.

[1]It may be of interest to add that in the protocol of the session of June 11, 1892, there is found a note which is not mentioned by Schleich in his memoirs. It is in the form of a letter addressed by Schleich to von Bardeleben, in which he says that when he was refused the floor after the reading of his paper, he was deprived of the opportunity to dispel a misunderstanding. He did not presume to sit in judgment on the differing opinions of his colleagues concerning the use of chloroform; he was intending to express his own opinion, only. But, he maintained, as regarded his own patients, that he must replace the dangerous chloroform anesthesia with his own safe method wherever it was possible so to do.

During the years which have followed Schleich's original presentation, his method has become the daily help of surgeon and patient. The essence of Schleich's contribution lies not simply in the idea of fluid infiltration. Halstead had shown, in 1885, what could be done in this manner with distilled water, but the publication consisted simply of a short letter to the editor of the *New York Medical Journal* (September 19, 1885) and it escaped notice on this account. Schleich's contribution was to show the superiority of isotonic salt solution as a medium, on the one hand; on the other, the effectiveness of such weak solutions of cocaine, therein, as had until that time not been credited with usefulness. Upon these two factors, however, the development of all that is now known as "Regional Anesthesia" has rested. To any one who has had experience with regional anesthesia on a considerable scale, Schleich's contribution must be regarded as of great and abiding significance. The use of this method in latter days for the reduction of fresh fractures was enough to establish its great importance—even though it had done nothing else than make it possible to avoid general narcosis in the reduction of recent fractures of the femoral neck, we owe Schleich a great debt. And yet, in a treatise as comprehensive as Labat's work on regional anesthesia, we find only an insignificant allusion to Schleich who so richly deserves credit in this field.

If it be said that Schleich's manner of presenting his valuable contribution has been the reason for the delayed and utterly inadequate recognition by the profession, I am aware that the statement may well be challenged. At the same time, as one who was an eyewitness to the lamentable episode, and as one who has had neither a personal acquaintance with the man, nor any personal interest in him, this is my opinion. The story of the incident is offered as presenting a moral which is too obvious for further comment.

Who hath given man speech? or who has set therein
A thorn for peril and a snare for sin?
For in the word his life is and his breath,
And in the word his death.
(Swinburne, *Atalanta in Calydon*, Ode IV.)

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

INFREQUENTLY books appear that mark epochs in the advancement of medicine. The assertion is ventured that MacEachern's book *Hospital Organization and Management*[1] is one of these. While many excellent hospitals served the public previously, the last 25 years have witnessed the general evolution of the hospital into a highly organized institution for the care of the sick and the advancement of medicine. In this movement Dr. MacEachern has held a prominent place. His service as a hospital superintendent and later as an official of the American College of Surgeons and other national organizations in the United States and Canada dealing with hospitals, supplemented by his surveys of hospitals in Australia and New Zealand, has given him a comprehension of hospital problems and a grasp of the details of administration beyond that of any other individual. He has analyzed, marshalled, and compressed this information into readable form and supplemented it with a wealth of practical details in this admirable volume which touches every part of hospital life.

Any individual, group, or community contemplating the organization and building of a hospital will avoid innumerable mistakes and find constructive advice in the chapter dealing with "Promoting and Building the New Hospital," from preliminary organization, surveys of the need, raising of funds, choice of architect, planning the building, contracts, to check lists of furniture and non-technical equipment.

The director, the various committees of the governing board, and the women's auxiliary will find their duties outlined and the methods of administering them detailed. The admitting officers will find proper procedure outlined and forms ready made for their use.

The medical staff and interns will be interested in the discussion of their functions and organization with helpful suggestions as to conference, consultations, autopsies, the duties of various committees, and their relationships to the patient, the hospital, and the public. Detailed information is given as to the various departments of medicine, surgery, orthopedics, obstetrics, ophthalmology, otolaryngology, and other clinical specialties with charts for records, check lists for medical and surgical supplies, and diagrams for the advantageous arrangement of units. The adjunct diagnostic and therapeutic departments such as laboratories, X-ray, physical therapy, and pharmacy will more clearly understand their duties and be helped in their organization and administration. Sample charts for records and reports, and check lists of equipment presented make this chapter invaluable.

The nurses will be interested in the valuable information concerning their education and the care of patients detailed in the chapters dealing with their problems. The superintendent of nurses will find her questions as to the organization and administration of her school and hospital answered in detail with all the forms and orders she may need outlined and check lists of all kinds of supplies furnished. The surgical and obstetrical nurses will be saved much time and effort by the procedures outlined and the check lists of supplies and instruments. They will also find comprehensive directions for the proper preparation and sterilization of instruments, supplies, and solutions in addition to a general discussion of their duties and responsibilities.

The same helpful suggestions with a wealth of detail will be found in the chapters dealing with the dietary, out patient, social service, medical records departments, and all the supplementary service departments such as business, purchasing, housekeeping, laundry, and maintenance. Indeed, one is amazed at the wealth of practical information which makes this book an essential in every department of the hospital.

That the cultural and social relations of the hospital are not neglected are evidenced by the chapters on ethics, public education, and the pervading atmosphere of service and ideals one finds everywhere throughout the volume.

This is a book that will live as a permanent contribution to medical and hospital literature. It is educational to laymen, helpful to hospital boards, and invaluable to hospital superintendents and personnel not only for the betterment of hospital organization and management, but also for rendering more adequate the scientific care furnished to patients. It is doubtful if one could ask a reasonable question pertaining to hospitals that could not be adequately answered in the pages of Dr. MacEachern's scholarly work.

A. B. K.

[1] HOSPITAL ORGANIZATION AND MANAGEMENT. By Malcolm T. MacEachern, M.D., C.M., D.Sc., F.A.C.P., F.A.C.H.A. Chicago, Illinois: Physicians' Record Co., 1935.

CERTAINLY few men are as well qualified to write on the treatment of fractures as Dr. Lorenz Boehler,[1] of Vienna. As director of the Hospital for

[1] THE TREATMENT OF FRACTURES. By Dr. Lorenz Boehler. 4th Eng. ed. translated from the 4th enl. and rev. German ed., by Ernest W. Hey Groves, M.S., M.D., F.R.C.S. Baltimore: William Wood & Co., 1935.

Accidents in that city, he is in a position to treat or supervise the treatment of all the common, as well as the bizarre injuries to which bones and joints are heir. He has gathered material representing 19 years of practice in traumatic surgery, and in 560 pages, augmented by 1,069 clear illustrations and many groups of statistics, has presented his thoughts and experiences in this subject. Ernest W. Hey Groves, of Bristol, England, has translated the work from the original German into English and has done so in a succinct, if not staccato, manner that is practically devoid of any redundancy. The style is not flowing but terse and to the point, and it is doubtful if more information (whether or not it is acceptable to all readers) could be had in the given space. The book is approximately 7 by 11 inches in size and is bound in blue cloth. The paper, printing, and reproductions of illustrations leave nothing to be desired.

The book is divided into three parts. Part I, entitled "General" takes up the broad subjects of after-treatment, apparatus, and dressings, application of plaster, treatment of compound and infected fractures, and also deals with operative treatment and the subject of non-union. In general the practice of the author in the management of these phases of fracture work conforms to the methods current in this country. Naturally, however, there are personal variations with which many of our authorities could not concur. As an example, one might mention the treatment of the open wound in a compound fracture with tincture of iodine, and the infiltration of the tissue contiguous with the contaminated field with local anesthetic.

Part II, entitled "Special Fractures and Dislocations" deals with the entire skeletal system beginning with the skull and ending with the phalanges. In this large group, of course, many controversial grounds are entered, and, going over these points critically, one might not always agree that the author's method is the one of choice in all parallel situations. However, in his treatment of these subjects Boehler speaks freely of methods that he has tried and discarded as wanting, and statistical tables offer valid reasons for their abandonment.

The third part of the book is composed of four subtitles appearing under "Appendices." These are (A) statistics; (B) the knee joint and its movements; (C) the position of the forefoot in flat foot, club foot and claw foot; (D) fractures and the surgery of accidents in relation to their environment.

In almost all of the subjects treated the normal is used for comparison, both in text and in illustration, and the requisites for the treatment of each condition are clearly outlined. Common mistakes in the case of each fracture are also enumerated. One experienced in this work might possibly resent the didactic manner in which treatment and mistakes are outlined, but for those who see fractures only occasionally it is probably an apt thought.

The chapter on the os calcis, rewritten and enlarged as the result of recent follow-up data, should be cited for special merit. It is based on studies of the normal mechanics of this bone and its articulation. Practically all methods of closed reduction are explained and statistics presented to show their comparative value in the author's hands; open correction, either as the primary treatment or to rectify a poor result, is touched upon only in passing. It might be added that throughout the book as a whole little space is given to the actual open methods many times required in the treatment of fractures.

Organization of a clinic where such work is done, adequate assistance, and the possession of mechanical appliances are stressed. No bibliography is found, and but few citations of other work in this field are noted in the various chapters. It is decidedly one man's opinion, and of little value from an encyclopedic view. This statement is not to be construed as in any sense derogatory, for the opinion of a man with Boehler's background in this field is definitely of value.

The entire book is readable, logical, and honest, and to be recommended without reservation.

JAMES K. STACK.

IN a single volume of 1111 pages and 1169 illustrations, Hinman has so well organized and outlined the contents of his book[1] on urology that one is readily able to get required information with a minimum of effort. The arrangement of the material makes the book a valuable reference work for the general practitioner and surgeon as well as the urologist. The contents cover all phases of medical and surgical urology as it is understood today. Controversial questions are discussed, and, while the author gives his personal views, he does not dogmatically close the issue.

The first chapters on comparative anatomy, embryology, and physiology are outstanding and simplify certain clinical features which have sometimes been difficult to comprehend readily. An adequate discussion of endocrine activity in the development of the genito-urinary organs and their diseases is given, together with the modern conception of vitamins.

The subject of pain as a symptom is discussed in general. The various types of pain are described in detail and anatomical charts are used which give the scientific basis by which the causes of various obscure pains associated with genito-urinary diseases may be more easily recognized. An excellent discussion is given of anuria in all of its phases including types, causes, and treatment.

The clinical phase of urology logically begins with a detailed examination of the patient and the methods used are presented in outline. General and special features of examination are also included.

Under the headings "Anomalies," "Obstructions," "Infections," and "Lithiasis," general urological diseases are discussed in an original manner, which makes for better continuity of thought, especially when the book is used by medical students.

[1] PRINCIPLES AND PRACTICE OF UROLOGY. By Frank Hinman. Philadelphia and New York: W. B. Saunders Co., 1935.

Under the heading "Obstructions," the various types of chronic vesical neck obstructions are described in logical order and the various types of treatment—surgical, electrical, radiological, and roentgenological—are discussed.

Under "Infections," the social and medical problem of gonorrhea is covered in detail, and the various clinical and therapeutic aspects of the problem are treated in a comprehensive manner. The sections devoted to pathogenesis and pathology of the various non-specific kidney infections are worthy of mention as is the section on renal tuberculosis.

The subject of urinary lithiasis is presented in greater detail than would be expected in a single volume work, all factors bearing on etiology, diagnosis, and treatment of the various types being thoroughly covered. The treatment of renal stone, including prophylaxis, is worthy of special mention.

In special sections, injuries, tumors, and neurogenic influence of the urinary organs are fully discussed. The female urethra is given attention commensurate with its importance in modern urological practice. Recent studies, directing attention to the many lesions of this structure together with their medical and surgical management, are presented. A noteworthy feature is a complete and scientific discussion of eneuresis and urinary incontinence.

Hinman's book is a very complete, modern treatise on urology which should be of value not only to the general practitioner and urologist but as a reference work to the medical student. HARRY CULVER.

THE previous edition of Zondek's work[1] on the endocrine glands appeared in 1926. This third edition in 1935, translated into English, is unquestionably one of the most important additions to the library of endocrinology. It might be said that it is at present the most valuable single volume on this subject. It is less ponderous than an encyclopedic work; it is more extensive than an outline; it is more detailed and thorough than a popular presentation. One-fifth of the book is used for theoretical considerations and physiology. The hypotheses arranged are stimulating; the physiological review is rapid but adequate and contemporary. It is significant that English and American work is given full credit. The clinical studies composing the remainder of the volume are profusely illustrated with short case reports drawn from the author's long experience. Not enough space, however, is given to bio-assay methods in the clinic. In the future it is to be hoped that improvement of assay methods will put clinical endocrine diagnosis on an exact basis. At present, as this book unfortunately shows, diagnosis is largely by inference from body build and non-specific metabolic measurements. In this sense then, this is an old fashioned book but it does contain a large amount of valuable material presented in an informative manner. A classified alphabetical bibliography is appended. PAUL STARR.

[1] THE DISEASES OF THE ENDOCRINE GLANDS. By Herman Zondek, M.D. 3d ed. rev. and enl. Translated by Carl Prausnitz, M.D., M.R.C.S. (Eng.), L.R.C.P. (Lond.). Baltimore: William Wood & Co., 1935.

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Find—we will help you find—an associate, an assistant, a partner. His ability, his smiles, his willingness to do, his knowledge of modern ways, his consultations—would save your time and give you hours and days and weeks each year to do the things you want to do.

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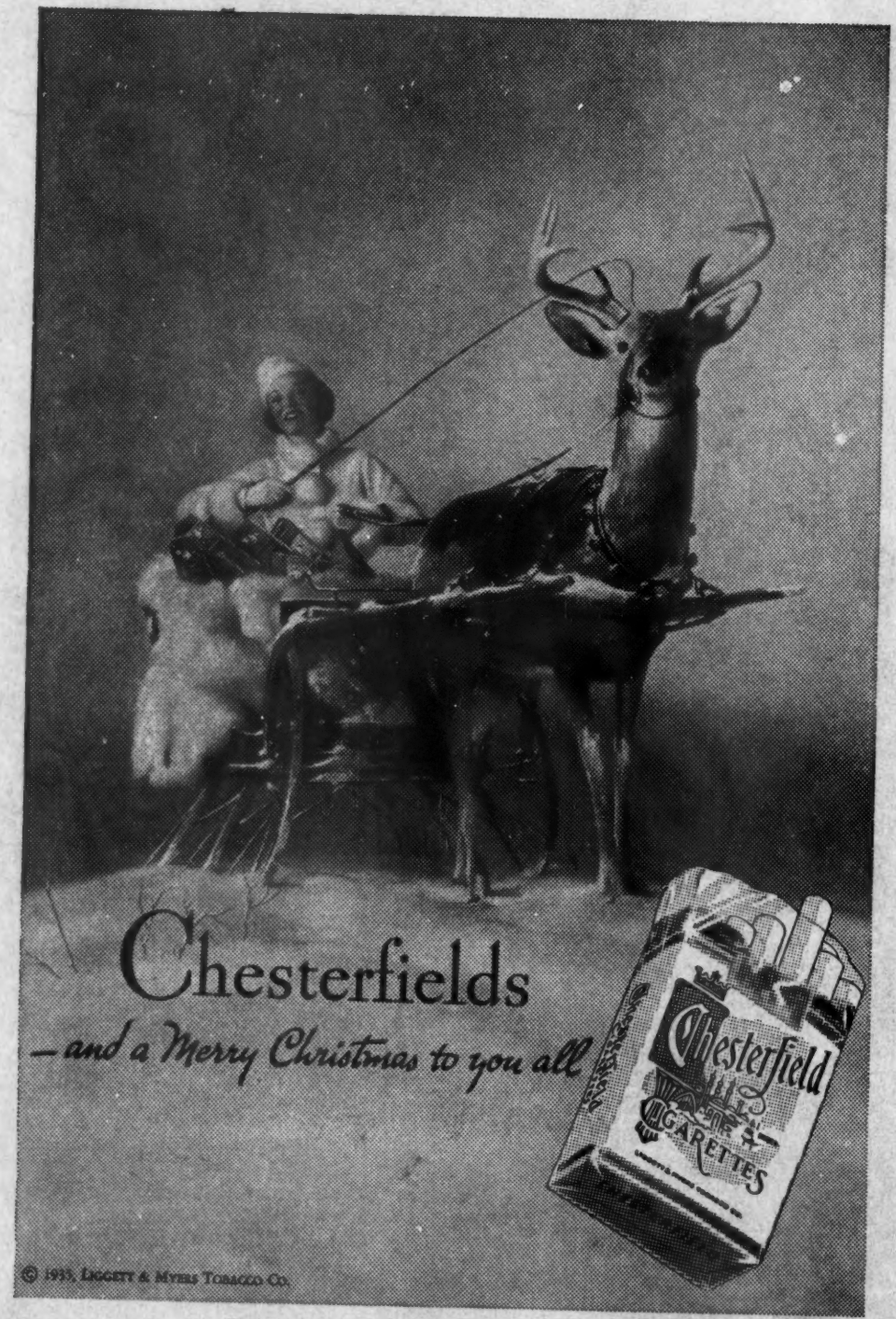
The National, State and Local Tuberculosis Associations of the United States



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